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LONDON SATURDAY AUGUST 21 1948

TUBERCLES OF THE CHOROID

BY

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AND

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[WITH COLOUR PLATE]

It has long been known that careful examination of the fundus oculi in cases of miliary tuberculosis and tuberculous meningitis may reveal tubercles of the choroid. The concentration of cases of this disease and the prolongation of life which has resulted from streptomycin therapy have provided the opportunity of studying these choroidal lesions and of observing their natural history. In this paper we discuss the frequency with which we were able to find tubercles of the choroid, describe their natural history, and report the findings of similar lesions in cases of tuberculosis in which there was no evidence of miliary spread or of meningitis.

Historical

It seems that Gueneau de Mussy (1830) was the first to recognize choroidal tubercles in miliary tuberculosis. Jaeger (1855) gave the first anatomical demonstration of these lesions. Manz (1858) described the detailed histology of a choroidal tubercle in a girl aged 15 who died of miliary tuberculosis. Fraenkel (1867) was the first to demonstrate them with the ophthalmoscope, though Von Graefe and Leber (1868) claimed that they were the first to demonstrate them with this instrument. They described the ophthalmoscopic appearance in detail. Cohnheim (1867) gave a detailed description of the necropsy on seven cases, and produced choroidal tubercles experimentally (with miliary tuberculosis) by injecting guinea-pigs with tuberculous material.

Various writers (Fraenkel, 1869; Weiss, 1877; Marple, 1912; Groenouw, 1920; Lotin, 1924) suggest that choroidal tubercles may develop in a few hours. This seems to be extremely unlikely, and the statement is almost certainly accounted for by incomplete initial examination. Bouchut (1869), Fraenkel (1869), and Weiss (1877) emphasized the value in diagnosis of finding choroidal tubercles. Hoeve (1925), Bollack *et al.* (1927), Mayrhofer (1935), and Baldenweck *et al.* (1938) describe cases in which the diagnosis of miliary tuberculosis was established by finding tubercles of the choroid. The literature was reviewed by Von Graefe and Leber (1868), Weiss (1877), Carpenter and Stephenson (1905), Bredeck (1916), Groenouw (1920), Bollack *et al.* (1927), and Paton (1932).

The frequency with which various writers found choroidal tubercles is tabulated below. In many papers it is very difficult to determine whether the cases referred to were meningitis only, miliary tuberculosis only, or a combination of these. The great majority were cases of miliary tuberculosis with or without meningitis. In cases referred to as meningitis only, the absence of post-mortem evidence that there was no miliary spread makes the accurate designation of these cases impossible: for, as mentioned below, careful histological examination not infrequently reveals miliary tuberculosis which on *x*-ray examination had not been evident.

TABLE I

	No. of Cases	No. of Cases in which Choroidal Tubercles were Found
Koch (1913)	60	8
Koplik (1907)	46	9
Garlick (1879)	26	1
Cohnheim (1867)	18	18
Groenouw (1920)*	225	54
Heinzel (1875)	41	0
Litten (1877)	52	39
Bredeck (1916)	11	2
Tooke (1915–16)	73	7
Moore (1922)	33	10
Carpenter and Stephenson (1905)	42	21
Marple (1912)	13	13
McDermott *et al.* (1947)	13	2
Debré *et al.* (1947)	84	22
Total	737	206 (28%)

* Quoting Stricker, 1876; Weissenfels, 1881; Bock, 1883; Money, 1883; Neskowic, 1884; Demme, 1888; Rheinhold, 1891; and Grüning, 1901.

A recent textbook (Doggart, 1947) merely states that "choroidal tubercles are visible in many cases of tuberculous meningitis."

The Clinical Material

Between January, 1947, and May, 1948, 65 cases of miliary tuberculosis and tuberculous meningitis in patients under the age of 14 were admitted to the Children's Hospital, Sheffield. Ten of these cases are excluded from the series to be discussed—four because the fundi were not examined, three on account of some doubt whether or not choroidal tubercles were seen, two because it was impossible to classify the cases owing to lack of *x*-ray and necropsy examination, and one because pneumococcal meningitis in a case of miliary tuberculosis masked the possible presence of tuberculous meningitis. The difficulty in some of these cases arose because the child was moribund on admission.

By the courtesy of Professor E. J. Wayne and Professor C. H. Stuart Harris we were able to examine the fundi of five unselected adults suffering from miliary tuberculosis or meningitis, and these are included in the series.

Four additional cases of pulmonary tuberculosis in children are discussed because of the finding of choroidal

tubercles without evidence of miliary spread or meningitis. These are not included in the series of 60 cases given in Table II.

Technique of Examination

It is obvious that the accurate examination of the fundi of infants and small children is a matter of considerable difficulty, and requires much patience and experience. The irritability occasioned by meningitis may make the examination particularly difficult. The pupils in all cases were dilated by 1% homatropine, and a sedative drug such as chloral, 1½ gr. (0.1 g.) per year of age, was used where necessary. The average time occupied for one examination of the fundi of a small child is about half an hour.

In almost all the children the fundi were examined repeatedly, but in view of the difficulties it is possible that in some cases choroidal tubercles were present but not detected. We feel, however, that in view of the care with which the examinations were carried out the number missed is likely to be small. It is certainly possible that histological examination would reveal tubercles which were too small to be detected by the ophthalmoscope.

TABLE II.—*Frequency of Choroidal Tubercles in the Series*

	No. of Cases	No. of Cases in which Choroidal Tubercles were Found
Miliary tuberculosis	14	7
" " with meningitis ..	28	18
Meningitis	18	1
Total	60	26

It will be seen from Table II that choroidal tubercles were found in 25 (60%) out of 42 cases of miliary tuberculosis with or without meningitis, and in only one case of meningitis in which there was no miliary spread. This last case was one of chronic tuberculous caries of the spine with terminal meningitis. Careful histological examination of the organs did not reveal miliary lesions. Of the five adults seen, all suffering from miliary tuberculosis with meningitis, choroidal tubercles were found in three.

That post-mortem examination, especially histological section, is necessary to exclude miliary spread is shown by the fact that in nine of our cases of meningitis in which *x*-ray examination failed to indicate miliary infection the necropsy revealed miliary lesions in the lungs, in six of these only on histological examination. Our figures for the cases of meningitis without miliary spread may not be quite accurate, as five of the 18 patients are still alive (though two have been observed for more than three months) and in three of the 13 fatal cases no post-mortem examination was possible. Therefore it may be that some of these eight cases should have been included in the group of cases of miliary tuberculosis with meningitis.

It is noteworthy that Sir Frederick Still (1927) said: "Tubercles of the choroid are rarely found in tuberculous meningitis if uncomplicated by miliary tuberculosis, whereas they are found in the majority of cases of miliary tuberculosis."

It is interesting that, whereas choroidal tubercles were found in 24 out of 33 cases in which there was *x*-ray evidence of miliary tuberculosis, they were found in only 1 out of 9 cases in which the miliary lesions in the lung were so small or non-opaque that they could only be discovered at necropsy. The significance of this observation is not clear.

The frequency of choroidal tubercles in the cases of miliary tuberculosis emphasizes the importance and value of a thorough examination of the fundi in all cases of possible miliary tuberculosis or meningitis. In some of the cases of meningitis in the series the finding of choroidal tubercles enabled a definite diagnosis to be made before an *x*-ray photograph of the chest was available and before a lumbar puncture had been performed.

In almost all cases the choroidal tubercles were found at the time of the first or second examination. Several writers have suggested that they are found only in the terminal phase of the illness. Our experience does not confirm that suggestion.

In only one case did we observe the appearance of choroidal tubercles during the course of streptomycin treatment. In view of the unsatisfactory response of some cases to streptomycin there seems to be no reason why this should not happen, but it must be remembered that the difficulty of full examination of the fundi of an infant makes it very easy to miss choroidal tubercles at the time of the first one or two examinations. Their discovery may well be explained by an inadequate search previously.

Description of the Choroidal Tubercle

In the early stage (Plate, Fig. 1) the choroidal tubercle appears as a rounded pale-yellow area with a matt surface and an indefinite edge merging into the red background of the choroid. It does not appear to be elevated. There may be only one tubercle, but usually there are several. The largest number observed by us in one eye was ten. The majority are one or two disk-breadths from the disk. The size is usually one-quarter to three-quarters that of the disk, but it is sometimes slightly larger than the disk. It is unconnected with the retinal vessels. It may be traversed by vessels, and if so they are apparently normal.

The histological appearance of the tubercle has been well described by many writers (Manz, 1858; Cohnheim, 1867; Bouchut, 1869; Weiss, 1877; Haab, 1879; Carpenter and Stephenson, 1905; Petrovic, 1926; Bollack *et al.*, 1927; Penman and Wolff, 1933; Tooke, 1936; Baldenweck *et al.*, 1938). Tooke (1936) draws attention to the absence of involvement of the vaginal space and optic nerve sheath, and considers that this is good evidence that choroidal tubercles arise from miliary spread and not by extension from meningitis.

There is swelling of the neighbouring choroid, with destruction of the stroma and infiltration with round cells, endothelial cells, and giant cells. Tubercle bacilli have been found in the lesions in large numbers. The retina is rarely involved, the tubercle being strictly limited to the choroid, but the retina may be thickened and show round-cell infiltration (Penman and Wolff, 1933). The retina is often involved by so-called conglomerate tubercles (Groenouw, 1920; Petrovic, 1926; Groenvall, 1933; Balza and Mosto, 1935; Harlowe, 1946).

The Natural History of the Choroidal Tubercle

We have found very little reference in the literature to changes in the choroidal tubercle. Von Graefe and Leber (1868), Fraenkel (1869), and Groenouw (1920) all mention the increase in size which can be observed by repeated observation. Cohnheim (1867), Von Graefe and Leber (1868), and Fraenkel (1869) describe the paling of the lesion which develops with its increase in size. Marple (1912) shows a good picture of an obviously old tubercle. Barlow (1910) says: "Choroidal tubercles may be observed in different stages of development in different parts of the fundus. They begin as minute round dots which gradually become more opaque in their centre." Very little reference has been made to pigmentation. Von Graefe and Leber (1868) mention the occurrence of pigmentation in large tubercles. Debré *et al.* (1947) say that "they gradually heal or disappear, leaving residual pigmentation only." They go

on to say that new choroidal tubercles may appear during the course of streptomycin therapy, and may serve as the only indication that all is not well. Lotin (1924) says that pigmentation sometimes occurs in these tubercles and that a chronic course is not uncommon in tubercles of the choroid.

The relative effectiveness of streptomycin treatment has enabled us to observe the changes which occur in choroidal tubercles over a period of several months. We have not seen any tubercles disappear during treatment, and in view of their pathology it would seem unlikely that this would happen. In some cases the tubercles may remain unchanged for some weeks. The possible prognostic significance of the absence of changes in the tubercles is mentioned below.

In at least 20 cases we observed well-defined and definite changes. The area occupied by the tubercle becomes larger and paler and the outline more distinct. Fine dots of pigment appear in and around the pale area (Fig. 2). This change is usually noted within eight weeks of starting treatment.

Following this intermediate stage the tubercle becomes paler still, until finally it becomes parchment-white, with a well-defined sharp edge, with heavy black pigment in and particularly around the lesion. There may also be a paling of the red choroid in the area immediately around the tubercle (Figs. 3–6). These changes may be complete in 12 to 14 weeks. We think that this parchment-white scar with a well-defined edge and heavy pigmentation may represent the healed tubercle. We have seen incomplete pictures of this several times but the final parchment-white stage in only five cases. One was a case of miliary tuberculosis with meningitis which responded well to four months' streptomycin treatment. The girl had a solitary choroidal tubercle, and we observed in it all the changes from the earliest to the final stage. She is now perfectly well, four months after the cessation of treatment. In another case, discussed below, we noted the same changes occur in a solitary choroidal tubercle in a girl with a tuberculous knee without miliary tuberculosis or meningitis. In another case one choroidal tubercle underwent these changes during the course of four months' streptomycin treatment (Fig. 3), while other tubercles, about a dozen in number, never progressed beyond the early intermediate stage: she died. In another case, admitted with a five-months history of miliary tuberculosis, one choroidal tubercle was found in this late stage, while others were found in the early and intermediate stages. The different age of these tubercles may well be due to a continued discharge of tubercle bacilli from the primary focus. In yet another case we observed a choroidal tubercle in the final stage in a child with a primary complex with no evidence of miliary infection or of meningitis. In this case the tubercle had not been observed in the earlier stages. This case is also discussed below.

Possible Prognostic Value of Changes in Choroidal Tubercles

We have seen too few cases and observed them for too short a period of time to be able to make any definite statement about the prognostic value of the changes we have described. But it is of note that the three patients whose choroidal tubercles changed to what we consider the final or healed stage, without other tubercles in earlier stages, are clinically well after fairly prolonged observation (all more than seven months); and that of the eight cases in which in spite of prolonged treatment (over three months) the tubercles failed to progress beyond the early intermediate stage death occurred in five and in the remaining three the response to treatment has been unsatisfactory and recovery seems to be very unlikely.

The following are records of two of the cases mentioned above. They illustrate the possible value of the changes described.

Case 1.—A girl aged 2 years 9 months was admitted with miliary tuberculosis. Four choroidal tubercles were seen in the early stage. She seemed to respond extremely well to streptomycin, and the *x*-ray picture of the chest became normal. But at the end of four and a half months' treatment, when the question of stopping the drug arose, the choroidal tubercles had not progressed beyond the early intermediate stage. The drug was therefore continued. Meningitis developed, and she died eight months after treatment had been started.

Case 2.—A boy aged 16 months was admitted with miliary tuberculosis. He seemed to respond very well to streptomycin, and the *x*-ray picture of the chest became clear; but his solitary choroidal tubercle failed to progress beyond the intermediate stage. Treatment was continued for six months—two months longer than the normal duration of treatment given here—and two months after the treatment was stopped tubercle bacilli were cultured from the gastric washings.

The appearance of a new choroidal tubercle after prolonged streptomycin treatment is naturally of bad prognostic significance. We have seen this happen only once.

Case 3.—A boy aged 15 months was treated with streptomycin on account of miliary tuberculosis with meningitis. One choroidal tubercle was found when he was first examined. In spite of four months' treatment this tubercle never progressed beyond the intermediate stage, and the radiograph of his chest showed only a partial clearing of the lung field. Five months after the treatment had been started a new choroidal tubercle was discovered. His fundi had been repeatedly and carefully examined, and it seems unlikely that this tubercle had been missed previously. The tubercle, furthermore, is in the early non-pigmented stage.

It is clear that a great deal more work is required on this subject, and we hope to give a further report in due course.

Choroidal Tubercles in Other Cases of Tuberculosis

It is generally considered that a tuberculous bacteriaemia is a common if not usual sequel of a primary tuberculous infection. It would seem possible that if careful search were made of the fundi of all children with primary tuberculosis an occasional choroidal tubercle would be found.

Randolph and Schmeisser (1915–16) stated that choroidal tubercles could be found, though infrequently, in practically any type of tuberculosis with a primary lesion. Brunthaler (1926) described a boy aged 4 with congenital syphilis and epituberculosis. Multiple choroidal tubercles were found. A year later the boy was in good health, and a few small rounded scars of the choroid were present. Weiss (1877) describes the finding of choroidal tubercles at necropsy in a man in whom no other evidence of tuberculosis could be discovered. Cohnheim (1867) said that choroidal tubercles could occasionally be found in cases of tuberculosis without miliary spread.

We have found choroidal tubercles in four such cases, but in two (thought by the physician and the radiologist to have merely a primary complex) the period of observation has at the time of writing been only a very short one (7 and 19 weeks respectively) and it is too soon to exclude miliary spread.

The two other cases, however, are of special interest, for they have both been observed for nine months and both are clinically and radiologically entirely normal. The following is a summary of these cases.

Case 4.—A girl aged 4 was first seen with a six-months history of pain in the right knee. Her tuberculin test was positive and a diagnosis of tuberculosis of the knee-joint was made. Three weeks after admission she was found to have a solitary choroidal

tubercle in the early stage. A radiograph of the chest showed a very doubtful generalized mottling of the lung field which the radiologist did not think was that of miliary spread. Tubercle bacilli were cultured from the gastric washings. Further radiographs were taken at intervals, and they have now been normal for four months or more and the child is well. She has not been given streptomycin or other specific therapy. The *x*-ray photographs are not reproduced in this article, because the mottling was so fine that it would certainly not show in a reproduction; the *x*-ray picture would probably have been passed as normal unless miliary tuberculosis had been particularly suspected. The interesting feature of this case is that we observed all the changes in the choroidal tubercle which we have seen in the streptomycin cases. The tubercle is now a parchment-white scar with heavy pigmentation around (Fig. 4).

Case 5.—A boy aged 3 was admitted on account of a tuberculous cervical lymph node. He had a history of recurrent cervical abscesses for two years. His tuberculin test was positive. A radiograph of his chest showed a picture identical with that of Case 3, and for the same reason the radiograph is not reproduced here. Tubercle bacilli were cultured from the gastric washings. A month after admission we found what we consider to be the final or healed stage of a choroidal tubercle (Fig. 6). The child is now perfectly well, and a radiograph of his chest has been clear for over four months. His choroidal tubercle remains unchanged. He too has had no specific treatment.

It is quite possible that both these cases are in fact examples of chronic miliary tuberculosis undergoing spontaneous cure, but in neither case was it possible to make that diagnosis from the radiograph of the chest.

Summary

Choroidal tubercles were found in 7 out of 14 cases of miliary tuberculosis without meningitis, and in 18 out of 28 cases of miliary tuberculosis with meningitis. In other words, they were found in 25 (60%) out of 42 of all the cases of miliary tuberculosis. They are therefore of great diagnostic value in these cases. They may establish a definite diagnosis before a radiograph of the chest or a lumbar puncture has been performed.

Choroidal tubercles were found in only one out of 18 cases of tuberculous meningitis without miliary spread.

In nine of our cases necropsy revealed miliary tubercles which had not been detected by *x*-ray photographs of the chest. It is emphasized, therefore, that in a case of tuberculous meningitis miliary spread can be excluded only at necropsy.

In no cases were choroidal tubercles found merely as a terminal phenomenon. They were found at the time of the first or second examination.

The natural history of the choroidal tubercle is described. As time passes, the margin of the tubercle becomes more definite, the tubercle enlarges, the centre becomes paler, and the tubercle and the surrounding area become stippled with pigment. In the final stage there is a parchment-white scar with heavy surrounding pigmentation and paling of the near-by choroid.

It is suggested that further observation may well show that the changes described are of prognostic value. Of those cases (eight in number) in which in spite of prolonged streptomycin treatment the tubercles failed to progress beyond the early intermediate stage, death has occurred in five and the remaining three are doing badly. All the cases in which the tubercle progressed to the parchment-white appearance without any tubercles in earlier stages were apparently cured.

Choroidal tubercles were found in four cases of tuberculosis in children in which there was no meningitis or certain evidence of miliary spread. In one of these cases a solitary choroidal tubercle was seen to pass through all the stages described above to the final or healed stage. It seems possible that careful examination of the fundi of all cases of primary tuberculosis will, in some, reveal choroidal tubercles.

Our thanks are due to Professor E. J. Wayne and Professor C. H. Stuart-Harris for allowing us to see their cases; to Drs. J. Burke, M. Powell, and J. Briggs for helping in the examination of fundi; to Mr. B. Nutt, ophthalmic surgeon, for advice; to Dr. J. L. Emery for the histology; and to Dr. T. Colver for letting us see his cases. The paintings are by Messrs. Hamblin.

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LEGENDS

FIG. 1.—Choroidal tubercle, early stage, on commencement of streptomycin treatment.

FIG. 2.—Very early choroidal tubercle at 3 o'clock. Several others in slightly later stages. Choroidal tubercles at intermediate stage at 7 and 11 o'clock. (After six weeks' streptomycin treatment.)

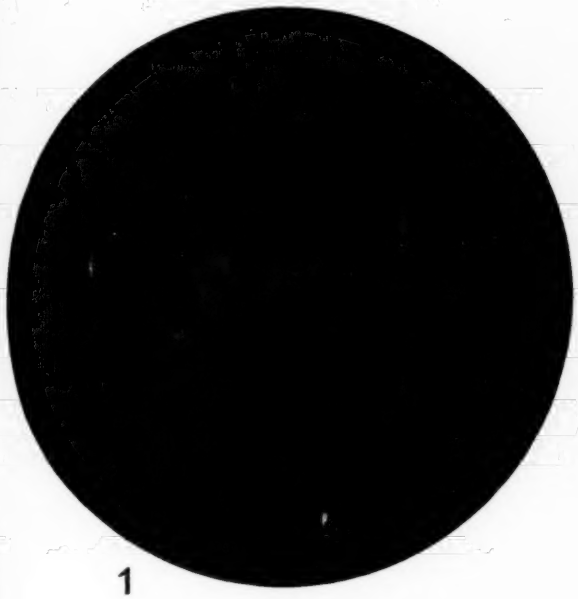
FIG. 3.—Late stage of choroidal tubercle at 3 o'clock, with two other tubercles in early intermediate stage. Case of miliary tuberculosis with meningitis which failed to respond to streptomycin.

FIG. 4.—"Healed" choroidal tubercle, Case 4, at end of six months' observation. Case of tuberculosis of knee without evidence of miliary infection. No specific treatment.

FIG. 5.—"Healed" choroidal tubercle. Case of miliary tuberculosis with meningitis on completion of streptomycin treatment, apparently cured.

FIG. 6.—Healed choroidal tubercle (Case 5). Case of primary tuberculosis without evidence of miliary spread or meningitis. No specific treatment.

R. S. ILLINGWORTH AND TREVOR WRIGHT: TUBERCLES OF THE CHOROID



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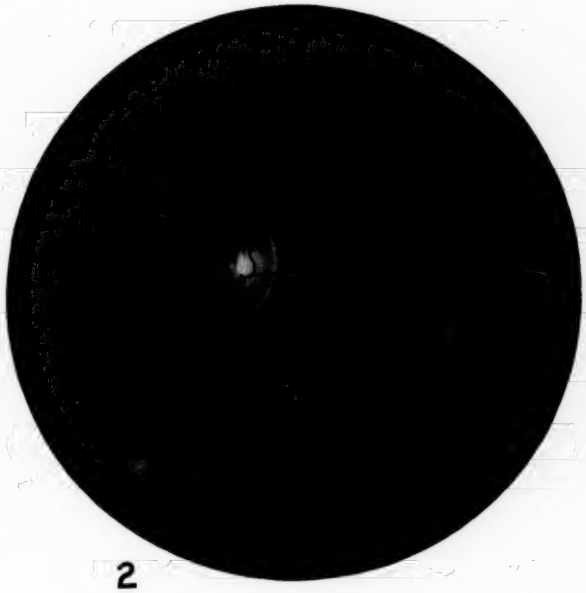

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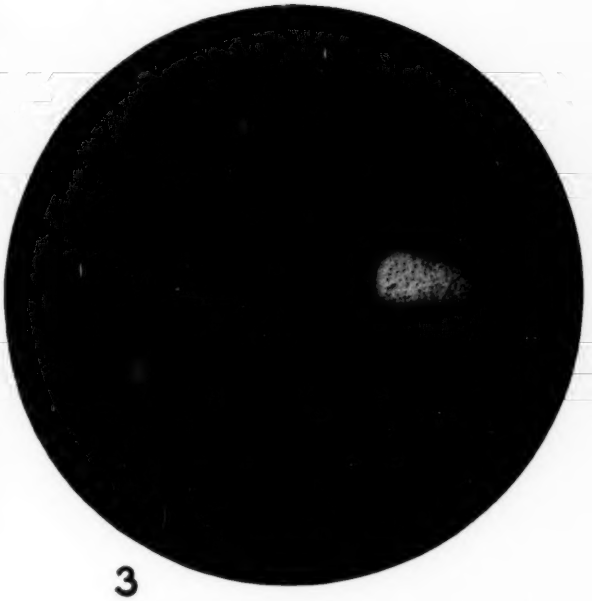

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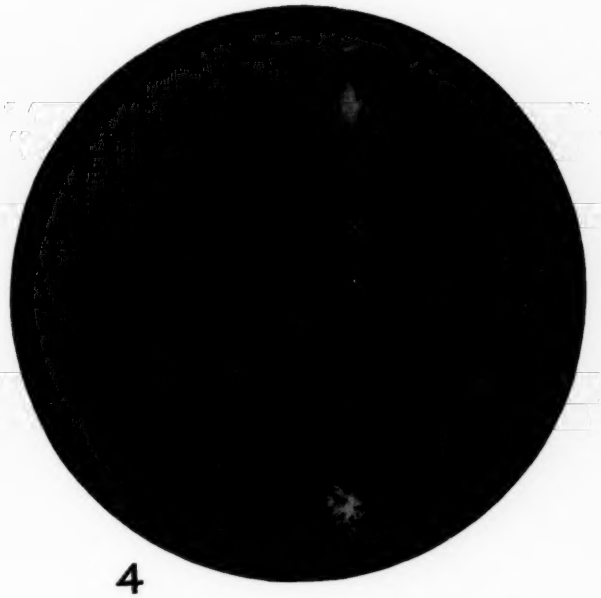

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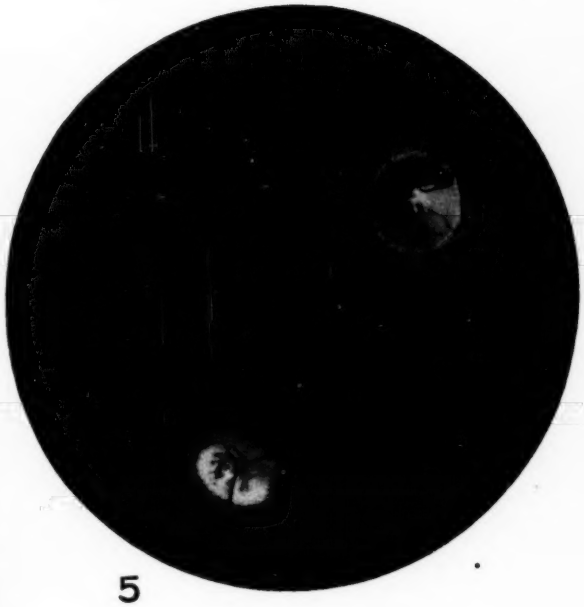

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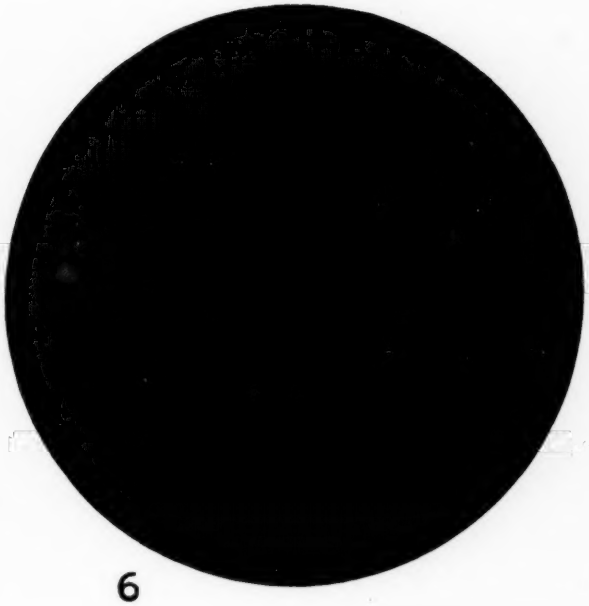

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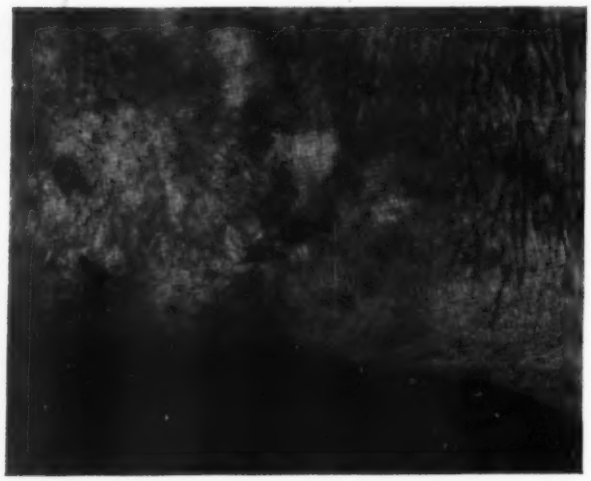

Fig. 1.—Shagreen skin of forearm. A. B., aged 52. Contact for 26 years. A "close-up" has been taken of the affected part to show the details of the skin degeneration.

Fig. 2.—Typical pitch wart on upper lip. C. D., aged 50. Contact for 4 years. Dome-shaped tumour with keratotic head proved innocent at biopsy. Two tiny warts near main lesion.

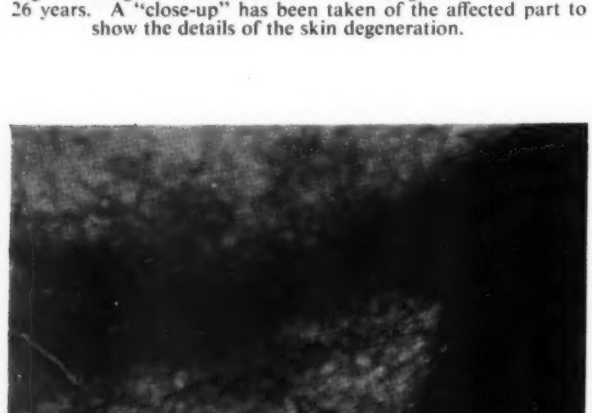

Fig. 3.—Pitch wart of the scrotum. E. F., aged 41. Contact with pitch and tar for 16 years. Tumour was innocent and disappeared with *x*-ray treatment.

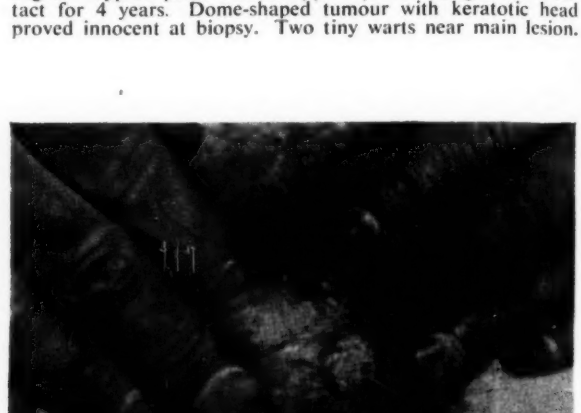

Fig. 4.—Epithelioma of scrotum. G. H., aged 42. Contact with pitch and tar for 17 years. Mushroom-shaped lesion had been growing for one month and had been mistaken for indolent boil.

Fig. 5.—Epithelioma of pinna. I. J., aged 39. Contact with pitch and tar for 20 years. He had had the lesion for one month and it was beginning to enlarge rapidly. Note similarity to papilloma in Fig. 2.

Fig. 6.—Epithelioma of upper arm. K. L., aged 49. Contact with pitch and tar for 34 years. Lesion for 5 weeks; treated as indolent boil. Note typical epithelioma. Slight break in wall of ulcer marks earlier biopsy. No glands. Healing perfect after *x*-ray treatment.

OCCUPATIONAL SKIN LESIONS DUE TO PITCH AND TAR

BY

PHILIP ROSS, M.D.

[WITH COLOUR PLATE]

The terms "pitch" and "tar" refer to a multitude of substances of varying composition and complexity, and a brief study of these compounds is desirable for the better appreciation of some of the problems they produce in industry. In addition to various asphalts and bitumens, Abraham (1945) in his recent treatise describes many types of pitch and tar, their origin and mode of preparation, but from our point of view we need consider only those derived from wood and coal.

Wood Tars and Pitches.—These are made by distilling various kinds of wood. Typical examples are the Stockholm, Norwegian, and Russian tars, all of which are used in the tarring of ropes, fishing-nets, boats, etc.

Coal-Tar and Pitch.—The main supply of tar in this country is from the destructive distillation of coal—a process carried out first and foremost in all gas-works, and, secondly, by the big iron and steel manufacturers. In the former instance we have the gas-works tar and in the latter the coke-oven variety. A small amount of blast-furnace tar is formed as a by-product in some of the Scottish iron-works where "splint" coal is used in the extraction of the pure metal from its ore; and an increasing quantity of tar is being made by the low-temperature carbonization of coal.

Chemistry

As the chemical composition of tar and pitch has a direct bearing on the production of skin lesions it is necessary to consider not only the mode of preparation but also some of the concomitant chemical changes. Tar is made by distilling coal, and pitch is the residue left after the distillation of tar.

Distillation of Coal.—At the gas-works coal is placed into retorts and heated to a temperature ranging from 1,200° to 1,400° C. (Martin, 1917), and, as the coal decomposes, ammonia, steam, coal-gas, and crude tar are evolved, leaving an amorphous mass of coke in the retorts. Similar decomposition occurs when coal is treated in the coke ovens, though the final products may vary somewhat from those produced in the retort houses. In both cases, however, the crude tar is collected for further distillation. In the low-temperature carbonization process the coal is distilled at a reading not exceeding 700° C.

Distillation of Crude Tar.—This process is carried out either at the gas-works by-products department or at the special tar distilleries. The results of a typical fractional distillation are indicated in Table I, but it must be stressed that the chemicals which are ultimately formed depend on several factors: (*a*) the type of coal in the original distillation; (*b*) the exact method of coal treatment—whether by vertical, inclined, or horizontal retort; (*c*) the temperature and conditions prevailing in the retort itself; and (*d*) the actual technique adopted in the tar distillation. The remaining pitch, which represents about a half of the original quantity of tar, is composed of carbon and numerous polycyclic hydrocarbons.

TABLE I.—*Distillation of Crude Coal-Tar*

Temperature	Compounds Produced
170° C.	Light oils: benzol, toluol, xylols
170–230° C.	Middle oils: phenols, cresols, naphthalene
230–270° C.	Creosote oils, tar oils
270–400° C.	Anthracene oils
	Residue: pitch

Note.—Numerous other compounds are formed in addition to the above: aliphatic hydrocarbons, pyridine, anilines, quinolines, sulphur compounds, etc.

Under normal working conditions tar is a black, heavy, viscous, and tenacious fluid, while pitch is a black, lustrous semi-solid. When subjected to an increasing temperature they become more and more labile, and tar begins to evolve fumes at about 80° C. The pitches fragment easily, and even the "softer" varieties may be ground to a fine dust when cooled to 10° C.

Industrial Hazards

Because of their unique properties and their cheapness tar and pitch have a widespread application in industry. Hazards from this source occur not only in the gas-works, coke ovens, blast furnaces, and tar distilleries, but also in the manufacture of producer gas, patent fuels, and "briquettes"; in asphalt-workers and road-repairers; in the making of certain electrical equipment such as cables, carbon brushes, carbon electrodes and insulations; in the lens industry, where pitch is used to imbed the lenses during polishing; in the building and allied trades, as in the preparation and the handling of roofing felt, special floor materials, paints, and weather-proofing compounds. Seamen and fishermen, too, handle a fair amount of tar and at times show typical lesions.

Pathology

When we consider the formidable array of coal-tar products, ranging from the light aliphatic and aromatic oils to the complex polycyclic hydrocarbons of pitch, it is not surprising to find that the pathological changes are both varied and bizarre. Our appreciation of these effects is assisted by the knowledge that the heavier oils, tar, and pitch have these characteristics in common: they are all skin irritants; are solvents of lipoids and cholesterol; are auxetic and keratoplastic; and, finally they are carcinogenic. Therefore a skin exposed to these materials may show evidence of epidermal and dermal inflammation; changes associated with an "eczematous" reaction; signs of excessive cell proliferation such as acanthosis, keratosis, and telangiectasis; and perhaps in the later stages malignant degeneration—usually a squamous-celled carcinoma.

An important physical reaction common to many polycyclic hydrocarbons is the property of fluorescence when their extracts are activated by ultra-violet light; and when these solutions are examined spectroscopically one can detect the carcinogens by their typical absorption bands (Mayneord, 1927).

Clinical Effects

Generally speaking, tar and pitch produce similar skin changes, but differences do occur by virtue of their respective physical and chemical properties. On the one hand, tar contains many light, middle, and heavy oils, while pitch is relatively richer in the polycyclic hydrocarbons. Furthermore, during the processing of pitch a fine dust can be generated with a high power of dissemination and penetration—a circumstance which explains the appearance of lesions (*a*) in remote parts of the body, and (*b*) in persons who work in the vicinity of, but not actually in, the particular workshop.

Regarding my own findings, I have been unable to differentiate between the effects of each material, as most of our employees are in contact with both pitch and tar either separately or mixed. Only rarely has it been possible to indict only one compound.

Tar Burns.—Owing to more thorough mechanization and enhanced methods of protection of the employees severe

tar burns are uncommon in industry. Should, however, the skin receive splashes of hot tar it must be cleansed either by swabbing gently with trichlorethylene or by the use of a special spray containing this solvent. The burn, usually of the second degree, may then be treated in the routine way. The belief that a tar burn could be followed rapidly by an epithelioma (Bang, 1923) is not substantiated by recent observation.

Tar Erythema.—This reaction is referred to in industry under a variety of names: pitch burn, tar "flashes," and "the smarts." It occurs in those in contact with pitch or tar fumes, and may appear either at the end of the day's work, when the employee emerges into the open, or during the working spell if he is engaged on outdoor duties such as loading or trucking pitch, or tarring roofs. The condition is apparently precipitated by sunlight or a strong wind, and is characterized by severe smarting of the exposed parts of the body, particularly the face and neck. The eyes, too, are often affected, resulting in a burning sensation and photophobia.

Though the symptoms may be sufficiently intolerable to compel the victim to seek refuge indoors, only rarely is he incapacitated for more than a day, and in the majority of cases he is quite able to resume work on the next shift.

In appearance tar erythema resembles ordinary sunburn, and may vary from a slight redness of the skin to a severe dermatitis with oedema. This latter reaction is now uncommon, and is to be seen in those working out of doors in bright sunlight. Of interest, too, is the fact that repeated attacks of this intense form of pitch burn is followed by persistent pigmentation. Conjunctivitis of a varying degree usually accompanies this condition and may necessitate the wearing of dark glasses temporarily.

The causal agent or agents are still unknown, but there appear to be two predominating factors—a sensitization of the skin by some ingredient of the pitch or tar fumes, and the action of bright sunlight on a skin thus rendered susceptible. With regard to the former, the chemicals indicted are thiazine, thiazone, acridine, and other azines present in tar (Leymann, 1930).

To prevent this condition it is necessary not only to apply the general precautionary measures outlined below (under "Prophylaxis") but to use in addition a protective paste or cream which is impervious to tar fumes, pitch dust, and light rays. After experimenting with several applications I have found a kaolin paste based on a formula suggested by Jenkins (personal communication) most efficacious when applied liberally to the exposed parts before each shift (see Appendix).

Treatment of the dermatitis itself should be carried out according to general principles, though I have found that the patients prefer cold cream or similar emollients.

Allergic Eczematous Dermatitis.—In my experience this reaction has occurred most infrequently. During the past twelve years I have carried out periodic and regular inspections of about 170 employees who handle pitch and tar, and my records show that during that time only six cases occurred, four of which were sufficiently severe to warrant a transfer to alternative work. This may appear somewhat surprising when we consider the virulent effects common to many coal-tar derivatives, such as the phenols, cresols, anilines, nitrotoluene, nitrobenzene, etc.

Folliculitis, Acne, Comedones.—These phenomena, though quite common among these workers, rarely cause symptoms or disability. The lesions are, in the main, limited to the forearms, face, and neck, and they pursue a chronic course, waxing and waning at intervals. As with ordinary acne, any severe secondary infection may result in the formation of small pitted scars, many of which can be seen on the faces and necks of some of our older employees. Of interest, too, is the presence in the scrotum and elsewhere of comedones from which one can express a small plug of sebaceous matter; and Henry (1946) has reported the presence of numerous sebaceous cysts in the scrotum of a man working in a low-temperature carbonization plant. These comedones are no doubt due to the mechanical obstruction of the glands by pitch dust.

In the selection of men for this type of work it is best to avoid engaging those who suffer from acne, as their condition is likely to be adversely affected by the materials they handle.

Pitch and Tar Melanosis.—This form of pigmentation has been recognized in industry for many years. Broadly speaking, there are two main types: (*a*) a fairly acute form where there is deep tanning of the exposed parts, especially the face, neck, and forearms, and (*b*) a more chronic variety accompanying other skin changes, such as keratosis and poikiloderma.

The first is a well-known event seen in those who work principally out of doors and who are in contact with pitch or tar fumes. The melanosis usually appears after a few months, is preceded by several bouts of tar "erythema," and persists throughout the summer months, with a tendency to fade during the winter or when the employee is transferred to indoor duties. An interesting concomitant phenomenon is the brown staining of the sclerotics. It is of interest to note also the presence of melanodermia in those in contact with creosote (Prosser White, 1934), anthracene (O'Donovan, 1929), and other coal-tar derivatives. The incidence of this form of pigmentation has been reduced considerably in recent years by the adoption of the various preventive measures directed against the development of pitch "burn."

The second variety of melanodermia evolves much more slowly and, in my experience, affects first the forearms and later the face, the neck, and occasionally the scrotum. After several years' contact with the materials in question one may perceive one of the following abnormalities: first, a brown mottling of the skin not unlike "freckles"; secondly, a diffuse dusky-red pigmentation; and thirdly, a patchy or reticulated deep reddish-brown discoloration. The two latter lesions either herald or accompany "shagreen" skin (Fig. 1).

The factors present in the causation of tar "erythema" appear to operate in the production of melanosis; for, according to Foerster and Schwartz (1939), who have carefully studied this latter condition, the tanning is due to the action of certain light rays on a skin made susceptible by the chemical photosensitizers in tar and pitch.

The mechanism of pigmentation has been investigated by Bloch (1927), who asserts that the epidermal dendritic cells of the basal layer of the skin and of the hair follicles (known as melanoblasts) produce the colourless precursor melanogen, which is transmuted into the pigment melanin by the action either of the ferment oxidase or of ultra-violet light. Some authorities believe that the chromatophores which normally carry pigment from the dendritic cells to the blood vessels may themselves generate melanin when affected by tar.

In contrast to this form of pigmentation is the observa- of Dr. R. E. W. Fisher (personal communication) that tar workers frequently show on their forearms pale areas of skin which are the relics of burns caused by splashes of hot tar.

"Shagreen" Skin.—This was the name given by Prosser White (1934) to a pathological state of the skin characterized by keratosis, pigmentation, dilatation of the superficial capillaries, and alteration of its texture. It can be caused by the action of a variety of agents such as excessive sunlight, *x* rays, and prolonged exposure to oils, pitch, and tar;

and clinically the condition may be regarded as a poikiloderma, several types of which, according to Kinnear (1935), have been described by the following authorities: Jacobi, Petges, Civatte, Riehl, and Hoffmann and Habermann. Only the condition reported by the last two observers had an occupational aetiology. The appearance of "shagreen" skin is unmistakable (Fig. 1), and on palpation the integument feels hard, rough, craggy, and inelastic. So far as pitch and tar are concerned one may perceive the inception of this degeneration after about ten years' close contact, when there appear patches of keratosis and scattered areas of telangiectasis on a background of dusky-red pigmentation affecting the forearms. As the condition advances the lesions become more marked and widespread, and involve also the dorsa of the hands, the face, and the neck, until in due course we have the full-fledged "shagreen" skin with pigmentation, telangiectasis, keratoses, areas of epidermal hypertrophy and atrophy, superficial fissuring, and possibly papillomata. Except for occasional irritation or burning sensation, symptoms are absent; and as the changes are permanent treatment is essentially preventive.

Neoplastic Changes

As has already been indicated, these are manifest as keratoses, papillomata, and epitheliomata.

(a) Keratosis

The earliest macroscopic evidence of the auxetic effects of tar or pitch is perhaps to be seen in the stratum corneum, where after several months of exposure one may detect small, raised flattened areas of keratosis ranging from 1 to 5 mm. in diameter on the dorsal surfaces of the hands, on the forearms, or on the face. These lesions, which closely resemble flat warts, usually remain unaltered for many years. On rare occasions the hypertrophy can be sufficiently prolific to result in the formation of a cutaneous horn.

(b) Papillomata

Papillomatous growths can occur in these workers after one year, though I have seen instances where they have appeared after only six months' contact. Three types have been observed: first, the simple wart; secondly, the typical pitch wart; and thirdly, the soft papilloma. The regions affected are the dorsa of the hands, the forearms, the face, the neck, and the scrotum. Only rarely do we see lesions on other parts of the body.

The *simple wart* resembles the common verruca, and may be sessile or pedunculated. It is innocuous and, as a rule, retrogresses and falls off—the latter process not infrequently being assisted by some minor trauma such as shaving. Should any prove uncomfortable or begin to enlarge rapidly, treatment by low-voltage *x* rays, by carbon dioxide snow, or by minor surgery should be instituted. Caustics are best avoided in these cases, as their irritant action might stimulate further proliferation.

The *typical pitch wart* appears as a small conical or dome-shaped tumour having a base of apparently normal skin and a keratotic head. The most common site is on the face, particularly near the nostrils (Fig. 2), and on palpation one finds the base hard and infiltrated. The lesion should be regarded as precancerous or frankly malignant, and should be treated either by low-voltage *x* rays or by local excision.

The *soft papillomata* are to be found in those who have been in contact with these substances for several years, and they appear on the face, especially on the eyelids and adjacent regions. They form small, soft, multiple, pedunculated tumours not unlike skin fibromata, and they are quite innocuous.

(c) Epitheliomata

Every worker who handles pitch or tar and anyone exposed to pitch dust is liable to become a victim of this hazard. As with the production of experimental cancer in mice there is always a variable latent period between the initial contact with these materials and the appearance of the growth—an interval which in my group of cases has extended from 18 months to 34 years. The occasional and casual handling of the compounds is scarcely sufficient to precipitate neoplasms, but five years' exposure to small amounts of pitch dust has resulted in malignant disease of the skin of the face. The cessation of all handling of these carcinogenic substances does not necessarily render the individual immune from further trouble, as growths have appeared after a "lag" period of many years.

Though pre-existing papules, keratoses, and follicles may suddenly become malignant the majority of these neoplasms are cancerous from the start, beginning usually as a typical pitch wart and enlarging to a diameter of 1/4 in. (0.63 cm.) or more within two weeks or so. The most common site for their appearance is on the face (Fig. 2), in the neighbourhood of the nostrils, but they also occur with some frequency on the dorsa of the hands, the forearms, the nape of the neck, the ears (Fig. 5), and the scrotum (Figs. 3 and 4). One patient, a man with an industrial history of 28 years' contact with pitch and tar who had advanced "shagreen" skin, suddenly developed four epitheliomata simultaneously—one on the back of the neck, another on the right ear, and one on each forearm.

If left untreated the tumour enlarges and the keratotic head falls off, leaving the classical epitheliomatous ulcer, circular in shape, with a hard infiltrated base, indurated edge, and raw bleeding surface (Fig. 6). At first the surface is covered with a tenacious slough giving the lesion the appearance of an indolent boil—an error which, in my experience, has occurred on two or three occasions. At times the base advances rapidly, and by a process of eversion converts the ulcer into a mushroom-shaped mass (Fig. 4). The growth is of low malignancy, its spread is slow and localized for quite a considerable time, and glandular involvement is very late. Histological examination reveals a squamous-celled carcinoma, and with early tumours the problem is not so much to find a malignant growth as a cancerous cell.

The best treatment is by the contact low-voltage *x*-ray apparatus giving about 4,000 to 6,000 r over a period of 8 to 14 days. The epithelioma becomes necrotic, sloughs away, and heals with a thin supple scar which is almost invisible. Early cancers often disappear after a single dose of 1,500 r.

Pathogenesis of Pitch and Tar Cancer

The Exciting Cause.—The association between occupation and epithelioma was first recorded by Pott, who in 1775, in his classical description of chimneysweep's cancer (or soot wart) of the scrotum, suggested that the soot (which contains about 40% of tar) was to blame. It was not, however, till the end of the nineteenth century that the lethal effects of pitch and tar were recognized, and in 1907 the definition "scrotal epithelioma occurring in chimney-sweeps and epitheliomatous cancer or ulceration of the skin occurring in the handling or use of pitch, tar, and tarry compounds" was added to the Workmen's Compensation Act (Henry, 1946).

Fresh light was thrown on the subject in 1915 when two Japanese research workers produced malignant skin growths in mice by painting their ears with coal-tar; and in 1924 Kennaway proved that a carcinogenic hydrocarbon was present in a fraction of coal-tar which distilled over a

temperature exceeding 500° C. Carrying this investigation further, Cook, Hewett, and Hieger (1933) were able to isolate from two tons of pitch a few grammes of a powerful cancer-producing agent which on spectrum analysis proved to be 3 : 4 benzpyrene—the now-recognized carcinogen of pitch and tar. In recent years numerous synthetic compounds having a similar action have been prepared—many of them related to the anthracenes (Cook, 1943)—and we now have about 300 such chemicals (Hieger, 1947).

Relative Potency of the Tars and Pitches.—It would appear, then, that the potency of a tar or pitch depends on the benzpyrene content, and any condition favourable to the appearance of this hydrocarbon would render its action more powerful. Such conditions are: (*a*) the particular type of coal that is mined in Britain and Belgium; (*b*) the distillation of this mineral at a high temperature; and (*c*) certain technical factors arising during the treatment both of the coal and of the tar. Generally speaking, gas-works tar and pitch are highly active; blast-works tar is believed to be less noxious, as are the pitch and tar derived from French coal; tar prepared during the low-temperature carbonization process is also relatively harmless, as is the tar obtained from the various woods; coke-oven tar and pitch prepared in America have low lethal effects (Cranch, personal communication); and the bitumens and asphalts, too, have a low carcinogenic action. Some observers, however, regard all types of tar and pitch with suspicion, and in any case from an industrial point of view it is almost impossible at present to obtain in sufficient quantities an inactive tar or pitch. Of the two, pitch is the more conducive to the development of malignant disease.

Ultra-violet Light.—The skin lesions poikiloderma and epithelioma caused by an excess of ultra-violet light resemble those produced by pitch and tar, but what the relationship is between the light rays and the polycyclic hydrocarbons is unknown. It is of interest to note, however, that the epitheliomata appear far more rapidly in rural districts than in urban—for example, in a country factory the latent period for a skin cancer is from 18 months to five years, whereas in London the analogous interval is from 9 to 34 years. It would appear, then, that an excess of ultra-violet light accelerates the action of the carcinogens in some mysterious way.

The "Lag" Period, or Delayed Action.—It has long been recognized that a malignant skin growth can appear several years after the individual has ceased all contact with pitch or tar. The longest interval in my series of cases is seven years, but Henry (1946) reports a "lag" period of 51 years in a patent-fuel worker. In what way the cutaneous cells have been "primed" is another puzzle to be solved.

Susceptibility.—There is no doubt that some workers are highly susceptible to the development of neoplasms of the skin, and this idiosyncrasy does not depend on the colouring alone, as blonds and brunettes are equally liable. Moreover, some employees who have well-marked shagreen skin have apparently never suffered from typical pitch warts, and there are others who have had numerous warts but never an epithelioma. On the other hand, an epithelioma was the first skin lesion (apart from attacks of tar erythema) to appear in seven of my cases. During the past 12 years, from a personnel averaging 170, including 10 women, I have noted skin growths in 102 individuals, and of these 16 showed malignant tumours. Among the women employees who come into contact with these compounds I have observed a corresponding ratio of papillomata but no incidence of malignancy. It is understandable why the exposed parts are affected, but why the scrotum too is involved is not easily explained. Contamination alone is not the answer, as I have never seen an industrial epithelioma of the penis or of the adjacent parts of the thigh.

The Increasing Incidence of Occupational Cancer

Since 1920, when epitheliomatous ulceration due to pitch and tar became an industrial disease notifiable to the Chief Inspector of Factories, there has been a steady increase in the number of cases notified. This is clearly shown in Tables II and III, taken from the Annual Report of the Chief Inspector of Factories for the years 1945 and 1946.

TABLE II.—*Notified Cases of Epitheliomatous Ulceration in Tar Distilling and Gas-works, 1921–45 (in Five-Yearly Periods)*

Period	Tar Distilling	Gas-works	Total
1921–5	67 (9)	22 (6)	89 (15)
1926–30	132 (12)	85 (43)	217 (55)
1931–5	178 (11)	88 (51)	266 (62)
1936–40	299 (10)	59 (19)	358 (29)
1941–5	358 (5)	91 (4)	449 (9)

Note.—The principal figures relate to cases, the figures in parentheses to deaths.

TABLE III.—*Extract from Table I of the Annual Report of the Chief Inspector of Factories for 1946*

Industrial Diseases

Epitheliomatous Ulceration	1920	1930	1940	1943	1944	1945	1946
1. Pitch	32	44 (1)	85 (1)	54	99 (1)	83	98 (3)
2. Tar	10 (1)	53 (9)	34 (3)	57 (1)	61 (2)	93 (1)	103 (4)

It is nevertheless encouraging to observe the decline during the past ten years in the number of deaths recorded, despite the somewhat alarming increase in the numbers notified.

What is the reason for the apparent increase in the incidence of industrial skin cancer? Several factors are involved: first, following the efforts of the Home Office Inspectorate, both the medical world and factory managements have been made aware of this particular hazard, and skin epitheliomata which were formerly thought to be a natural visitation are now being considered correctly in the light of occupational environment. Secondly, many workers have now passed the "latent" period and their skins are liable to develop this lesion. Thirdly, because of the increasing interest displayed by surgeons, pathologists, and industrial medical officers, many more biopsies are being performed, and tumours which were at one time thought to be innocent are now found to be malignant.

Prophylaxis

The widespread use of pitch and tar and the dangers attendant on their use render the question of prevention of the utmost importance in industry. This problem can be attacked in several ways.

1. Substitution

As gas-works tar and pitch are carcinogenic, attempts should be made to substitute other less noxious compounds—as, for example, wood tar, bitumen, tar from the low-temperature carbonization process, and fractions from petroleum. Unfortunately the application of these alternative products is not always practicable either because of their unsuitability or because of their prohibitive cost.

2. Protection of the Worker

One of the requirements of the Factories Act is that all injurious fumes and dust should be removed by mechanical means; and the strict observance of this regulation would go far in the prevention of skin troubles. Where practicable all the processes should be mechanized to avoid undue handling of the materials; machines should be enclosed, and efficient exhausts should be fitted to carry away any dust or fumes that are generated.

Employees should be made aware of the hazards involved, and be trained to work cleanly and to prevent the dissemination of effluvia. Also they should be encouraged to report early any abnormal skin condition or appearance.

The use of protective clothing, too, is of paramount importance. Overalls made of stout drill material or denim should fit snugly at the neck, wrists, and ankles, and they should be changed and cleaned daily. I would recommend also a daily change of underclothing; but this, unfortunately, is not feasible while clothes rationing remains so severe. The use of "any old suit" which rapidly becomes soiled and impregnated with pitch and tar is to be condemned.

Gauntlet gloves made of stout rubber or leather, when used judiciously in certain processes, have a definite protective value, as have suitable goggles in shielding the eyes.

Employees should be encouraged to maintain a high standard of body cleanliness, and time and facilities should be provided so as to ensure a wash and shower bath after every shift. The application of "barrier creams" and the plentiful supply of warm soft water should obviate the use of special cleansers—either chemical or abrasive—which of themselves may be a fruitful source of dermatitis. The restricted supply of soap is causing concern and the use of substitutes is not always desirable, though "teepol" or similar "wetting" agents may help to eke out the soap ration.

In some firms it has been the custom among those working with pitch, tar, and heavy oils to use soda ash for washing purposes, and some of the older employees are unequivocal in their assertion that the use of this alkali prevented the development of warts. The use of caustic soda has also been referred to by O'Donovan (1929) in the popular treatment of skin growths by men working with tar. Though weak alkali solutions may be beneficial, the use of them cannot be recommended, because of the serious risk of dermatitis.

I am of the opinion that barrier creams fulfil a useful function, and at our works several types are available, depending not only on the materials handled but on personal idiosyncrasy. In addition to the usual properties of a protective cream, the compound should be opaque to ultra-violet light. Several chemical and cosmetic firms now specialize in these preparations and are prepared to make suitable applications, especially if they are supplied with samples of the materials used and provided they are given information about the conditions under which these are handled—for example, the temperature of the process and the use of ancillary substances and solvents. The creams should be applied liberally to the hands, forearms, face, and neck before each working shift.

In a recent investigation by Kennaway (personal communication) it was shown that the use of a protective cream on the skin of a mouse delayed considerably the appearance of experimental tar growths.

3. Selection

While it is not yet possible to foretell those likely to develop skin lesions I nevertheless examine all prospective candidates for this type of work, and I exclude those with existing skin disease or with a bad dermatological history. The question of skin colouring is in my opinion not very helpful.

4. Inspection

I regard the frequent and regular inspection of employees an important step in prophylaxis. Not only am I able to detect early skin changes of which the worker himself may be unaware, but I am able also to educate and reassure him, and discuss with him the various precautionary measures that are being adopted. I see almost every pitch and tar worker at three-monthly intervals, and those who have or have had skin lesions are examined as often as their condition warrants. Only in this way can we be sure of instituting early treatment and prevent serious complications.

5. Propaganda

Most industrial concerns are loath to broadcast indiscriminately the hazards obtaining in the use of these compounds, and do not favour the distribution of leaflets or the posting of warning notices (except the one recommended by the tar distillers), as these measures are likely to deter men from working in these processes. Instead, individual education and training are left to the medical officer and to the foremen and charge hands. In this way adequate welfare supervision is ensured without creating unnecessary alarm.

Statistical Surveys

As medical officer to a large engineering firm who manufacture various types of carbon products, I have had during the past 12 years the opportunity of observing some 170 employees who have handled pitch and tar more or less consistently during that period. My investigation has been facilitated by the small labour turnover, for at the end of 1947 there were 87 workers with at least 12 years' service to their credit and 53 with 20 years' or more.

An analysis of the incidence of their lesions is shown in the following tables. In Table IV I have recorded the number of personnel who have shown the appropriate condition for the first time—subsequent recurrences being ignored; and in Table V are noted the latent periods of the various manifestations. Tables VI and VII show the sites of election for papillomata and epitheliomata.

TABLE IV.—*Incidence of Lesions Among a Personnel (including 10 Women) Averaging 170 from 1936 to 1947, Arranged in Four 3-Yearly Periods. Recurrences Not Included.*

Lesions	1936–8	1939–41	1942–4	1945–7	Total
Allergic dermatitis	2	2	2	0	6
Folliculitis, acne, comedones ..	15	14	21	16	66
Melanosis	10	7	5	3	25
Shagreen skin	17	6	2	1	26
Keratosis	9	8	11	3	31
Papilloma	18	24	40	20	102
Epithelioma	2	2	8	4	16

The following points are worthy of note:

1. *1936 to 1938.*—I began my investigations during these years, and while collating the data I encountered conditions which had been in existence for several years. This was particularly the case with the 17 instances of shagreen skin, which had developed several years before 1936.

2. *1942 to 1944.*—The increased incidence of folliculitis, keratosis, papillomata, and epitheliomata during this period can be accounted for in two ways: first, the advent of war necessitated an augmented production of accessories derived from pitch and tar, and from 1940 onwards not only were greater amounts of these materials being handled but working hours were extended. Secondly, the establishment of a dispersal factory in a rural area brought a further 20 men into contact with these compounds from the years 1941 and 1942. By taking into account the latent periods one can appreciate the rise in the number of lesions recorded during these years.

3. *1945 to 1947.*—I attribute the reduction in skin troubles in this interval to the intensification of the campaign of prophylaxis, especially in the provision of clean overalls daily, in the more widespread use of barrier creams, in the supply of soft water for washing, and in the allowance of ample washing-time.

We are indebted to the medical inspectors of factories for their assistance in the clean-overall service and to a firm of cosmetic manufacturers for their research work on suitable creams.

4. *Papillomata.*—Of the 102 cases 62 showed multiple warts.

5. Epitheliomata.—16 men developed malignant skin tumours (all confirmed by biopsy), and of these 9 had previously suffered from simple warts and 3 had multiple epitheliomata. There were no fatal cases. The 9 with simple warts have also been included under "papillomata."

TABLE V.—*Latent Periods*

Folliculitis, acne, comedones	.. ..	1 month or more
Melanosis	.. ..	6 months to 5 years
Shagreen skin	.. ..	Over 10 years
Keratosis	.. ..	6 months or more
Papilloma	.. ..	6 months to 43 years
Epithelioma { Rural district	.. ..	18 months to 5 years
Epithelioma { Urban district	.. ..	9 to 34 years

Note.—The shorter latent period for the appearance of a skin carcinoma in the rural district is discussed under "Ultra-violet Light." Because of this latent period all employees who leave the firm are requested (where practicable) to report to us any rash, pimples, or warts that might appear at some future date.

TABLE VI.—*Site Incidence: Papillomata*

Employees affected ..	102 (including 6 women. Age from 29 to 66)
Single lesions	40
Multiple lesions ..	62
Site affected:	
Nose and lips	43
Cheeks, eyelids, chin ..	24
Ears	6
Neck	8
Scalp	1
Hands	29
Forearms	16
Scrotum	11
Leg	1
Anus	1
Total	140

It would appear that the site of election for these growths is the face (48%); the incidence on the hands and forearms is 32%, and on the scrotum 8%.

TABLE VII.—*Site Incidence: Epitheliomata*

Employees affected ..	16 (no women. Age from 34 to 62)
Single lesions	13
Multiple lesions ..	3
Site affected:	
Cheeks	5
Nostrils, lips	4
Ears	3
Neck	2
Scalp	1
Forearms	3
Arm	1
Scrotum	4
Total ..	23

The most frequent site for the appearance of epithelioma is on the head and neck regions (65%). The upper limbs and the scrotum each show a rate of 17%.

Summary

A brief account has been given of the preparation, the chemistry, and the industrial uses of coal-tar and pitch.

The following clinical effects have been described and discused: tar burns; tar erythema; allergic eczematous dermatitis; folliculitis, acne, and comedones; melanosis; shagreen skin; neoplastic changes, and epitheliomata.

The pathogenesis of pitch and tar cancer has been discussed.

Various prophylactic measures have been described.

A statistical survey has been included.

APPENDIX

Formula for Starch and Kaolin Paste for the Prevention of Tar Erythema

Starch	28 g.
Kaolin	1,600 g.
Phenol	36 g.
Glycerin	50 ml.
Distilled water	2 litres

The starch is made into a thin paste with about 50 ml. of water and poured into 950 ml. of boiling water. The boiling is continued for a few minutes to ensure the breaking up of the starch cells. The phenol and glycerin are added to the mucilage, and finally the kaolin, in small quantities at a time, with constant stirring. The final mixing is best done with the hands to make certain that no lumps remain.

The phenol prevents the development of mould in the paste and the glycerin renders the paste more "supple" and prevents rapid drying and cracking. Colouring matter such as cochineal may be added for a better cosmetic effect.

I wish to thank the Controller of H.M. Stationery Office for permission to reproduce the tables from the Annual Reports of the Chief Inspector of Factories, and Professor Kennaway for permission to publish the results of his experiment with a barrier cream in the production of experimental skin cancer (1946). I also wish to express my indebtedness to Mr. A. E. Wiggs, B.Sc., Dr. Henry MacCormac, Dr. Davidson, late H.M. Inspector of Factories, and Dr. L. B. Bourne for their kind assistance and suggestions.

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OBSERVATIONS ON TUBERCULOUS MENINGITIS IN CHILDHOOD*

WITH SPECIAL REFERENCE TO EARLY DIAGNOSIS

BY

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Of all the clinical pictures met with in children there is probably none which has sustained such intense interest over so many years as that of tuberculous meningitis. The first account of the clinical course of the condition was given over 180 years ago by Robert Whytt during the time that he occupied the chair of medicine at Edinburgh University. His description, under the title "Observations on the Dropsy in the Brain," is still recognized as a classic.

Interest in the subject of tuberculosis in childhood, and of tuberculous meningitis in particular, has received added stimulus from the trials being given to streptomycin therapy. If this new form of therapy is to receive reasonable trial patients submitted to treatment must be diagnosed early. This constitutes something of a challenge to clinicians, because it must be admitted that we have not advanced greatly from the days of Whytt in the early diagnosis of tuberculous meningitis, no matter how improved are our methods of investigation in relation to the later stages of the condition.

Successive Clinical Phases

It is customary to describe the clinical course of tuberculous meningitis in terms of three stages. Sir Frederick Still used to say that while no fool could err when the middle and last stage had been reached, the wisest physician might fail to recognize that anything serious was amiss with a child in the first and early stage. It is this early

*Summary of an address given to the Harrogate Medical Society on Dec. 11, 1947.

stage with which I am concerned, but in order to present things in their proper perspective I will first briefly describe the clinical course of a typical case of tuberculous meningitis in childhood from its insidious onset to its tragic termination.

Because I am anxious to lay stress on clinical aspects I propose to consider my subject in the light of what I feel to be the immediate problem faced by the family doctor who has not the advantages of laboratory facilities at his elbow.

First Stage

This is usually described as *the prodromal or the premonitory stage*. There are many recorded opinions concerning the average length of this period. While most are agreed that it usually lasts for some two to three weeks it has been recognized that it may last for as long as two months. It is generally agreed that there are no absolutely characteristic symptoms during this phase of the disease. Indeed, a disconcerting feature is that not only are symptoms rather vague but they are too apt to resemble those often met with in a number of other conditions. To add to the worry of the clinician, when tuberculous meningitis is suspected at this early stage the problem is not merely one of differential diagnosis as between a number of serious possibilities, but is equally one of deciding whether the symptoms are in fact attributable to a serious or to a comparatively minor condition.

Most of us can remember children whom we have had under observation on account of anorexia, fretfulness, or lassitude, and possibly also failure to gain weight, and who in the course of only a week or two have developed headache and constipation severe and persistent enough seriously to raise the question of meningitis. If vomiting is also present we would instinctively suspect meningitis, but it has to be remembered that cases occur in which the vomiting is so infrequent or is so isolated in character that its significance may not be immediately grasped. Nevertheless I have no hesitation in saying that of the triad vomiting is unquestionably the earliest symptom in the great majority of cases no matter how occasionally it may occur in the individual case. A further point well worth mentioning is that sometimes a child not complaining of headache will complain of pain or perhaps only discomfort in the neck—a symptom never to be lightly ignored.

I like to look upon this stage as one characterized by symptoms which individually appear to have no serious import but collectively reflect considerable and to some extent increasing physical and emotional debility, and concerning which the most striking feature is their persistence.

Second Stage

The prodromal stage is usually described as being followed by *the period of irritation* or, as it is sometimes called, the stage of invasion. The transition from one to the other is only gradual. Despite the fact that such suggestive terms as "invasion" and "irritation" are employed, the point to remember is that even in this second stage many of the pronounced signs and symptoms commonly associated with other forms of meningitis are much more likely to be absent than present. This is certainly the case until such time as the stage of irritation is in process of passing over to the third and terminal stage—that of increased intracranial pressure.

In saying this I do not wish to suggest that the clinical picture in the second stage is not often typical to the extent of being almost diagnostic. It is during the second stage that prodromal symptoms lose some of their earlier vagueness and assume more pronounced form. A disgruntled fretful child is seen to become irritable to the point of petulance. Lassitude gradually merges into drowsiness. As drowsiness increases the breathing tends to become irregular, and even before this the child can be heard every now and again—sometimes at long, sometimes at short intervals—to give a prolonged, deep sigh. Even at this stage older children may be found up and about in their homes, but more often than not they are curled up in a chair in a shaded corner of the room, their faces turned from the light. If, on the other hand, the child is in bed all the points I have mentioned can be observed without turning down the clothes. Often the cheeks are slightly flushed although there may be little or no fever. The so-called tache cérébrale can be easily obtained. In many cases you will find the child grinding his teeth, sometimes for hours on end. In strange contrast the eyes retain an unnatural brightness.

The moment you draw down the clothes the child shows his resentment. He pushes away the examining hands, clutches at the sheets, protests in a high-pitched voice, firmly closes his eyes, and remains curled up on his side, his legs flexed at the hips and knees. The likelihood is that the bones of the thoracic skeleton will be a little prominent, and that the abdomen from being a little full is beginning to show a scaphoid contour. Increasing anaemia becomes apparent. There are periods of great restlessness during which the child picks away at his lips, the umbilicus, or genitalia; and may cry out, not in pain but rather irrationally. These periods are only too likely to alternate with times when the patient may sit up and take an interest in his immediate surroundings. Often there is a frown of the forehead, which is the more striking because it is so rare in children. These intermissions of spontaneous interest gradually lessen in length and in frequency as weakness increases.

When you come to examine the child in greater detail the pulse will be found to be slow and possibly irregular until eventually it usually assumes the characteristics of the so-called cerebral pulse. Too much should not be expected from examination of the central nervous system at this stage. It is the exception rather than the rule to elicit Kernig's sign—and although many disagree I find little help from Brudzinski's sign. An extensor plantar reflex is sometimes present in older children, but its absence is of little or no significance. Nor is neck rigidity by any means a constant finding. There are, on the other hand, cases which show evidence of what I like to describe as objection rather than rigidity in response to flexion of the neck. This I realize is only a matter of degree; nevertheless, I have found that sensing neck objection of this sort has helped me to anticipate the development of other and more conclusive signs.

If the child with tuberculous meningitis is an infant the fontanelle provides valuable clinical evidence. Here again, however, it is, I am convinced, well worth while trying to recognize differing degrees of departure from normal in an attempt to arrive at an early diagnosis. There can of course be no mistaking the tense pulsating fontanelle, but before this stage is reached it is sometimes possible to appreciate a fullness characterized by "bogginess" rather than by tension. Where this occurs in the presence of other suggestive findings it is of undoubted value in arriving at a diagnosis. Even where there is neck rigidity, head retraction of any degree is unusual, and where it is present a noticeably thick exudate in the medullary region is usually found at necropsy.

In general, then, it can be said that true neck rigidity, head retraction, and Kernig's sign, as they are found in other forms of meningitis in childhood, are by no means typical where infection is due to the tubercle bacillus. A more common finding at this stage is the sudden appearance of motor palsy, albeit slight, in some form or another. Ptosis, unilateral or bilateral, and strabismus, usually internal, are the most common. Either of these may be associated with irregularity or inequality of the pupils. More rarely, but not altogether uncommonly, a limb or the facial muscles are involved, sometimes temporarily for some days, only to reappear.

So much for the stage of irritation, which usually lasts for a week but which more often than is sometimes stated may last longer than that.

Third Stage

I do not propose to enlarge upon the terminal stage. Suffice it to say that during this stage the slow pulse gives place to a rapid pulse, coma develops from drowsiness, and the temperature from being moderately irregular rises progressively. There may be delirium; convulsions are common—so also is incontinence of urine and faeces; flushing is pronounced; and wasting is increasingly rapid. The gaunt facies, the dry lips, the haggard expression, and the rattle of the throat make for one of the most trying ordeals parents at the bedside of a dying child have to face. I have dwelt on this because I feel that in striving to improve our knowledge of the condition we

should be mindful of the anguish that we may be able to save parents to whom at present we can offer little comfort, only understanding.

Such, then, is the unfolding clinical picture presented by tuberculous meningitis in childhood. This picture contains three salutary warnings, one related to each phase of the picture. Briefly these lessons are: (1) The clinical picture in the third and terminal stage is so predominantly one of intracranial disease that sight is apt to be lost of the fact that infection of the meninges has been secondary to an earlier infection elsewhere in the body; (2) The clinical picture in the second stage is sufficiently typical to suggest a correct diagnosis, but it is not dependent for its characteristics on findings associated with other forms of meningitis in childhood; (3) The symptoms which go to make the clinical picture of the first or prodromal stage have four more or less constant characteristics—namely, vagueness, insidiousness, a slow progressiveness, and a certain ominous persistence when reviewed over a period of time.

The Problem of Early Diagnosis

It is to this baffling prodromal period that I shall devote the rest of this address. When we are faced with the problem of diagnosis in a child presenting vague but rather persistent symptoms, what factors should or should not raise the question of tuberculous meningitis?

And here I would say at the outset that I think we would succeed in diagnosing tuberculous meningitis earlier were we, in the first place at least, to consider whether active tuberculous infection is present rather than whether there is or is not meningitis. In saying this I am influenced by two things. First, it is recognized that there is a special risk of meningitis occurring during the three months which succeed primary infection; and, secondly, the symptoms of primary infection, of early miliary infection, and of early meningeal involvement are vague and indefinite. The symptoms, consisting of nothing more than slight fever in a child who for some unexplained reason has gone off his food or off his feet, may pass unrecognized.

I have already referred to the importance of headache, constipation, and vomiting in the early symptomatology. Judged by the literature on the subject there is no agreement on which of these three is the earliest. As I have said, in my experience vomiting has almost always been the first. A point of some importance, more especially in the case of older children, is that mention of vomiting is often omitted in the mother's description and that reliable information is got only if leading questions are asked. The reason for this is that the vomiting bears no relation to food, has no periodicity, and occurs at long intervals. A point concerning the constipation is that more often than not it is associated with a clean tongue. In most cases the headache is frontal, but in the early stages at least it is not usually severe enough to keep the child awake.

Quite apart from actual wakefulness at night, restlessness may be an early complaint. Sometimes by restlessness at night a mother means irregular and unusual breathing during sleep; and observations which I made on cases of early tuberculous meningitis when holding resident hospital appointments certainly did suggest that irregular breathing in sleep is by no means uncommon at an early stage in these cases. Other forms of restlessness in sleep have been given a good deal of prominence in the literature but I have not found them to be constant findings. More commonly a mother comments, without any suggestion of worry, that her child is more difficult to waken in the morning. This may mean little, but if later the mother remarks that her child is not only difficult to waken in the morning but has gone to sleep after tea or has voluntarily expressed a wish to go to bed earlier than usual, then the clinician must be very much on the alert.

While on the question of information given by the mother there is another aspect which I should like to stress. Important as questions and replies may be, very often it is the aside made by the mother or her casual observation that sounds a note of warning. The mother is the first to note changes in temperament in her child. Because she is perplexed she is not always good at describing what she has noticed. If a mother talks about her girl or boy "being different" or "not himself" it is always worth while probing deeper. Disinclination to play is an early sign of many illnesses in childhood. But on many occasions it has struck me that this temperamental change or adjustment goes a stage further in cases of tuberculous infection. The child not only limits his physical and mental activities but, over and above that, withdraws from contact with others and retires within himself. He wishes neither to be spoken to nor to talk. He shows a preference for a chair in the corner. He is moody to the point of sullenness; and then, for no apparent reason and with altogether surprising suddenness, recovers some but not all of his former vivacity and interests. A certain sense of tiredness, perhaps even weariness, persists and increases as the moods come and go, but still the eyes remain bright and the pupils a little dilated. I do not wish to suggest that the picture I have drawn is diagnostic, but I do say that in the presence of other indefinite symptoms this fitful temperamentalism in association with tiredness is an important feature of most cases.

Physical Signs

And what of physical appearance and physical findings as distinct from emotional behaviour? There is no typical appearance. While it is true that many children show evidence of previous disease or of debility of varying degrees, many others are apparently robust, well clad, and rosy-cheeked. In the individual case an appearance of ill-health is of value in diagnosis, but the fact that a child looks physically healthy and does not seem to have lost weight in no way offsets the possible significance of other symptoms suggestive of tuberculous infection. On the other hand, no matter what my own assessment of a case may be, I never ignore a mother's suspicion, much less her statement, that a child has got thinner. And when I have compared my own observations with those of parents I have usually found that any discrepancies have been due to the latter sensing an early almost infinitesimal loss of subcutaneous tissue as yet unaccompanied by wasting of muscles.

Another feature worth noting is the tendency of many of these patients—including those of apparently robust physique—to show an unnatural and early dryness of the skin. This is usually most in evidence over the trunk, but sometimes it first appears over the inner aspects of the thighs. Although of itself a small point, I mention this minor observation as being one of many which go to make up the elusive clinical picture of some cases of early tuberculous meningitis.

There are of course other aspects of a case which may legitimately influence diagnosis. Chief among these is a history of exposure to infection or the presence of tuberculosis elsewhere in the body. So far as exposure is concerned there is, I think, a tendency to jump to conclusions too readily. It is not the fact that a member of the household has had open tuberculosis that matters, but the fact that the patient has or has not been in contact with the suspected person at a material time (Table I). My experience on this particular point is mainly based upon hospital work in Edinburgh and to a smaller extent in the south, but it has always surprised me how very often

TABLE I.—*Environmental Factors**

1. *History of contact in connexion with cases due to human infection or to infection of unknown type:*

No. of cases lacking reliable history of contact	48
No. of cases with no reliable history of contact but with history of tuberculosis in family	42
No. of cases established as having been exposed at a material time to contact with:	
A. Unsuspected open infection in:	
a. A parent	20
b. A sibling	5
c. A grandparent	4
d. Other relative	9
e. A member of the household (or children's community) not a relative	13
f. Neighbours	6
B. Previously suspected infection in a relative:	
a. Discharged the Services	4
b. On leave from sanatorium	2
c. Awaiting transfer to sanatorium	7

2. *History concerning milk consumption in 45 cases known to be due to bovine infection:*

Particulars Concerning Milk	No. of Cases from	
	Rural Area	Urban Area
No information	5	9
Never boiled	13	5
Occasionally boiled	3	4
"Pasteurized," "T.T.," or "invariably boiled"	2	4

* The cases included in the series were drawn from many administrative areas in both Scotland and England.

careful inquiry can establish that there has been an unquestionable risk of exposure. Another finding which has impressed me is the frequency with which there is a history of previous children in the family having died from tuberculosis. In the series on which I am basing my remarks this was true of no fewer than 17 children, representing over 8% of all the cases. Equally striking in this same series is the fact that no fewer than 25 (12%) of the 205 cases occurred in first children who at the time of their death were "only" children. Why this should be I am uncertain. Nor can I explain why it is that so often this dread form of disease seems to pick out "the bonniest of bairns."

Tuberculin Skin Test

Where tuberculous infection is known to exist elsewhere in the body the occurrence of new symptoms, no matter how vague or slight, should at least bring to mind the possibility of meningeal involvement. Even in those cases in which infection may have been previously suspected and not established, recourse should in such circumstances be had to radiological examination without delay. The fact remains, however, that in a large proportion of children known to be suffering from intracranial tuberculosis it is not possible to establish during life the presence of infection elsewhere in the body. I would suggest that this common experience has its lesson. We should accept that we cannot expect physical signs to help us very much where early meningitis is suspected. That being so, much greater use should be made of tuberculin skin tests to determine at an early stage whether or not a child has in fact been infected and acquired sensitivity. It may be argued that these tests are apt to fail in cases of tuberculous meningitis. That is true, but in my experience only rarely, and I am yet to be convinced that it ever fails in the early stages of the condition. In part the explanation lies in the fact that severe anaemia, impaired peripheral circulation, hyperpyrexia, and cachexia, which may contribute to failure or suppression of skin reactions, are not present in the early stages of tuberculous meningitis. Subject to the employment of a reliable method I have always looked to tuberculin skin-testing for invaluable help. While the Mantoux test is the most reliable method, I prefer the multiple puncture test for use in the home as being simpler and almost as reliable as the Mantoux and because it is unquestionably more reliable than the jelly or patch test. To get a negative reaction to a reliable test in a vague worrying case can, by allowing a more favourable prognosis at an earlier stage, save parents as well as physician much anxiety. A positive reaction in the presence of signs or symptoms even vaguely suggestive of meningeal involvement, more especially in a child of 6 years or under, should point to the necessity for immediate admission to hospital for further investigations.

Lumbar Puncture

If the tuberculin skin test is of value, what of lumbar puncture? In what I have said I have tried to keep the problems of the family doctor to the forefront, and the question of whether to perform a lumbar puncture or not often constitutes a problem in itself. It is always wise to study the parents before deciding to do a lumbar puncture in the home. There is the risk that in their distraction the relatives may attribute any subsequent developments to the puncture. And it is, I think, well to remember that in the early stage of tuberculous meningitis the findings in the fluid, while they may be valuable, are not likely to be conclusive. The fluid is always clear but not usually under pressure in the early stage. In the later stages a typical fluid shows a cellular increase mainly mononuclear in character, an increase in protein, a decrease in sugar, and a pronounced decrease in chlorides. It has been said that a chloride content of less than 650 mg. per 100 ml. is virtually diagnostic. This simplifies the position overmuch. A chloride content of 650 mg. is suggestive of tuberculous meningitis, but can in my experience occur where intracranial inflammation is attributable to organisms other than the tubercle bacillus.

In the early stages of tuberculous meningitis the cerebrospinal fluid findings vary considerably. There may or may not be an increase in the cell content. It is no uncommon finding for polymorphs to be as numerous as lymphocytes. In some cases the polymorphs are actually in excess. This is more likely to occur where the patient is a small child and where the condition is running a more than usually rapid course. Great differences are found in the protein content. A normal figure or one as high as 400 or 500 mg. per 100 ml. may be obtained.

A reduction in chlorides is the exception and not the rule in the early stages of tuberculous meningitis. In nine cases, each of which I had under continuous clinical observation for over five weeks and from each of which cerebrospinal fluid was obtained at intervals of not less than five days, early signs of irritation preceded any significant reduction of chloride content of the fluid. On the other hand, a low sugar content appeared some four weeks before death in four of these cases. Whereas the sugar content tended to fluctuate, the protein content, which rose comparatively early, continued to increase gradually and progressively to a level which varied for each case but which, once attained, was maintained until death.

It will be appreciated, therefore, that in the early days of tuberculous meningitis there is a certain element of chance in what one may find in the fluid. And where lumbar puncture is performed in the early stage the findings are likely to provide no more than equivocal information to be evaluated in relation to clinical observations.

There is just one reservation I must make in this connexion. If cerebrospinal fluid from a well-established case of tuberculous meningitis is allowed to stand, a "shimmery" frail coagulum appears in the course of a few hours. Sometimes this occurs in fluid obtained early in the course of the disease. On four occasions I have known this coagulum to appear during the prodromal stage. Allowing that the likelihood of this occurring so early is slight, it is, I think, worth looking for in any suspected case, because if found

it is so highly suggestive. The same may be said of choroidal tubercles and changes in the optic disks. These were encountered at an early stage in only five of my series, but were of value in allowing of more rapid diagnosis than would otherwise have been possible. Extensive miliary spread was demonstrated post mortem in all five cases.

The Factor of Age

And now I come to my last point, which I have left to the end because it has a bearing on almost all that I have already said. As is well known, the form which a particular disease assumes may differ in infancy as compared with older childhood. This tendency applies to tuberculous meningitis and in particular to the prodromal stage of the condition. A point of importance in diagnosis is that tuberculous meningitis is rare under 6 months of age. It occurs with greatest frequency in the second and third years of life (Table II). This is of some importance

Table II.—*Age Distribution*

Age Period	No. of Cases	Age Period	No. of Cases
6 months and less	6	7–8 years	6
6 months–1 year	31	8–9 ,,	8
1–2 years	45	9–10 ,,	3
2–3 ,,	37	10–11 ,,	5
3–4 ,,	18	11–12 ,,	4
4–5 ,,	16	12–13 ,,	1
5–6 ,,	11	13–14 ,,	2
6–7 ,,	10	14–15 ,,	2

because the condition tends to run a more rapid course in infancy that in older childhood. Of the triad—headache, vomiting, constipation—headache is rarely a recognizable feature in infants. It is sometimes said that headache is indicated by screaming and by the hands being raised to the head. This is contrary to my experience. My impression is that the infant with early tuberculous meningitis tends to be quiet rather than noisy. Often, indeed, it is unnatural drowsiness that first gives a hint of the presence of trouble. Most commonly, however, vomiting is the first symptom. Sometimes, and I think frequently, it is preceded by a sudden and complete loss of appetite. Almost invariably it is followed by constipation.

In contrast with the condition in older children the vomiting tends to be persistent rather than erratic, but if a careful history is taken it will usually be found that anorexia has preceded even the first vomit. As I said earlier, the fontanelle often raises suspicion at an early stage. So also does a certain strangeness in the expression—a strangeness usually described by the mother as vacant, but which to me sometimes suggests a sense of apprehension. Eventually the expression becomes fixed. Nor is there, I think, any question that convulsions are more likely to occur early in infants, whereas in older children they are usually a late rather than an early sign. It is rare to come across recognizable papilloedema in the small baby.

Another point worth mentioning is the frequency with which there is a history of recent ear discharge in cases of tuberculous meningitis in infants. My experience is that it occurs in some 20 to 30% of those under 3 years of age. There is always the risk that it may delay the real diagnosis. For what it is worth I might mention my impression that it is usually, if not only, in those cases of tuberculous meningitis with associated otitis that the child is seen holding his head or ears as if in pain.

Onset of Symptoms in Bizarre Form

So much, then, for the differences in infancy as compared with other ages. I need hardly say that we must always be prepared for the case which presents at the onset in an altogether unusual, even bizarre, form. I have twice had children brought to me on account of ptosis which had developed suddenly and which later proved to be the beginning of meningitis. In five other cases the first complaint was of paresis of a limb or part of a limb, or of the facial muscles. In two of these five cases the paresis disappeared only to be followed after an interval by the symptoms of early meningitis. In two cases difficulty in walking was the first thing noticed. In another child, brought on account of hysteria, a coarse intention tremor of one hand was observed in the out-patient department. Ten days later the child showed signs of early meningeal involvement. Where there is inflammatory involvement of the cervical cord the head may be held in any grotesque position, and I have twice had infants referred to me in which torticollis was the first outward evidence of tuberculous meningitis.

Bizarre symptoms lacking other explanation—and they include the precipitately occurring convulsion lacking apparent organic or reflex association and the sudden development of gross athetoid-like movements in older children—should always raise the question of tuberculous meningitis. So, I think, should a history of headache dating back to an accident some time previously. This may seem rather irrational, but there was a history of accident in no fewer than 7% of my cases. Even allowing that the connexion between accident and illness was more a figment of the mother's distraught imagination than a reality in some instances, the figure is too high to be ignored, and it has some pathological justification if Rich's theory concerning meningeal infection is accepted.

Lastly, importance should always be attached to a child's past health history. Analysing my records, my impression is that the point of significance is not so much whether the child has had measles or whooping-cough recently as whether he has or has not had a number of debilitating conditions, more especially if they have followed in rapid succession or have been of pronounced severity.

Robert Whytt concludes his classic with a note on "The Cure of a Dropsy in the Brain." In this note he says: "If this disease could be known early, and before any considerable quantity of water has been collected, it might probably sometimes be cured." He goes on to say: "As it never discovers itself till so much water is accumulated . . . as to disturb the action of the brain we have little to hope from any medicine."

Whytt thought in terms of "purgatives, diuretics, blisters, frictions, exercise, and diet." To-day we are at the stage of giving trial to streptomycin, which may provide us with some of the answers we seek. As yet we cannot say, but success in the use of streptomycin, whether for research purposes or as a form of therapy, will, to quote Whytt again, depend on "knowing the disease early."

The United Kingdom Trade Commissioner at Delhi has forwarded to the Board of Trade the following report of an answer to a question in the Constituent Assembly of India (Legislative). "The country is self-sufficient in regard to the common vaccines used for public health purposes, such as cholera vaccine, T.A.B. vaccine, plague vaccine, and vaccine lymph. The indigenous production of other drugs is inadequate to meet the country's requirements, and the hospitals obtain Indian-made medicines to the extent they are available. The question of increasing the production of certain essential drugs is under the consideration of the Government of India in the Ministry of Industry and Supply in consultation with the Ministry of Health. In view of the shortage of foreign currency the Government are already following a restrictive policy in respect of imports of medicines and drugs, including patent medicines. Drugs are allowed to be imported only if they are essential."

STAINLESS STEEL WIRE FOR CLOSING ABDOMINAL INCISIONS AND FOR REPAIR OF HERNIAE

BY

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Stainless steel wire was introduced for the closure of abdominal incisions by Babcock in 1934. It has been adopted with success in other clinics, notably the Cleveland Clinic, where Jones, Newell, and Brubaker reported favourably on its use in 1941.

"One of the first clinical impressions of alloy steel wire used alone without stay sutures in the closure of abdominal wounds was the frequency of satisfactory healing, regardless of such adverse factors as carcinomatosis, deep jaundice, and general cachexia."

They confirmed the findings of previous authors (Dambrin; Kaufmann, Johnson, and Lesser) that the wire is well tolerated by the tissues, that it can be used in the presence of infection, that it produces solid closure, and that eviscerations are rare. When the wound is infected a residual sinus seldom occurs. The incidence of infection in clean wounds is markedly reduced.

Problem of Abdominal Disruption

We consider that the elimination of post-operative disruption is one of the most compelling factors in choosing stainless steel wire rather than catgut, silk, or thread in the closure of abdominal incisions. It is well recognized that disruption is relatively common and that when it does occur it is a very serious matter. Jones *et al.* (1941) reported 44 disruptions in 4,154 consecutive laparotomies not closed with steel (1.05%) with 15 fatalities (34% mortality); Meleney and Howes (1934) reported on 50 cases of abdominal disruption which occurred at the Presbyterian Hospital, New York, an incidence of about 1% of all their abdominal operations, with 22 deaths, a mortality of 44%. Relatively few of the latter cases were suffering from malignant disease, though it is well known that this is one of the most important factors predisposing to disruption (White, 1934).

Abdominal disruption is due to one or more of the following causes: (1) failure of the biological processes of wound healing; (2) interference with healing, or the dissolution of recently healed tissues, by a local infection or by digestive enzymes; (3) a disruptive force such as vomiting, coughing, abdominal distension, hiccuping, and sneezing.

(1) The strength of a wound correctly closed with stainless steel wire is unaffected by time, and is sufficient to withstand more than normal strains. It should therefore be used when there is any evidence of dietary deficiency, or where there is any factor present such as malignant disease or jaundice.

(2) Wound infection accelerates the dissolution of catgut and also prevents normal healing from taking place. The same is true of wounds bathed with biliary, pancreatic, or other digestive ferments. If non-absorbable ligatures such as thread and silk are used infection penetrates into the interstices between the filaments of the material and cannot be thrown off by the body until the ligatures have themselves been discharged. Stainless steel wire is a single inert filament offering no concealment or retreat for micro-organisms. No matter how serious an infection may be present or occur, granulation tissue grows on the surface of the wire, closing in over it without any residual pocket. The wire is heat sterilized, and there is no risk of it introducing infection.

(3) The strength of stainless steel wire is such that no disruptive force can burst a wound closed in one of the manners suggested. This certainty of strength is a great comfort to surgeons, assistants, and nursing staff. Come what may, the risk of abdominal disruption can be ignored. Confidence in the abdominal closure is easily transmitted to the patient, who is thus more likely to co-operate willingly in getting out of bed within or about 24 hours after the abdominal operation.

Ten Years' Experience

Our own experience dates from 1938, when we started using the wire for closing all major abdominal laparotomy incisions, especially in the presence of potential infection and in cases of malignant disease. The method was so uniformly successful that we adopted it almost as a routine Soon we began using it for large incisional herniae which would have been very difficult to repair by any other method. The complete success attendant on these bad cases encouraged us to extend the use of wire to all herniae requiring reinforcement of the defect. Finally, one of us recently had occasion to close with wire an abdominal incision in the other. This enables us categorically to state that the patient is not aware of the presence of the wire.

We have used stainless steel wire in more than 2,500 major abdominal incisions and in a large number of minor cases in old or frail patients. There has been no instance of abdominal disruption or post-operative herniation. In a large proportion of the major cases the abdomen was opened for malignant disease. The patients have on the whole been seriously debilitated, and have provided a stern test of the methods here described.

It is too early to judge of hernial recurrences, because most of these repairs (there have been 300 so far) have been done during the last four years. We hope later to be able to have a sufficient number of cases followed up for proper assessment of results. So far there has been no recurrence.

FIG. 1.—Photomicrograph of human abdominal wall 21 days after operation, to show absence of inflammation around stainless steel stitch and good deposition of fibrous tissue. Compare the catgut suture.

The Wire.—We use stainless-steel alloy wire, standard wire gauge 31 or 32 for abdominal incisions and large incisional herniae, 33 or 34 for inguinal and femoral herniae. It is strong, supple, smooth, and not ionizable in the body fluids. Knots can easily be tied with it and are secure. The tissues react to its presence by the formation of fibrous tissue only. We have demonstrated histologically in post-mortem material that there is no inflammatory or foreign-body response (Fig. 1). The wire, strong in itself, acts as a framework over which the body deposits a dense mat of fibrous tissue over a

narrow zone. The wire may or may not fragment after months or years, but the scar does not stretch.

Organisms cannot penetrate the smooth surface of the wire. It therefore does not act as an impediment to the healing of wounds which are infected at the time of operation, or which have become infected later. On occasions a sinus has formed (e.g., following operations such as the closure of a colostomy with a pericolostomy hernia), but this sinus has in our experience healed spontaneously. We have known of only one to persist for more than a week or two. This patient was a diabetic with a colostomy and three difficult herniae.

There are advantages to be gained from its use other than those due to its inherent qualities. Stay sutures, with their concomitant discomfort, liability to infection, and ladder scars, are never necessary. Our custom is to close skin incisions with Michel's clips, thus eliminating punctures through which infection may burrow from the surface. This is of particular advantage where there is risk of faecal contamination from an adjacent colostomy.

In dealing with hernial orifices we are of the opinion that stainless steel is the suture material of choice. No special instruments and no set of preconstructed filigrees are required. The method is immediately adaptable to the closure of an orifice of any size or shape. In fact the patient is given a "made-to-measure filigree." The security of the closure renders bed rest not only unnecessary but undesirable. The usual stay in hospital after herniorrhaphy is seven to ten days, after appendicectomy four to eight days, after gastrectomy 12 to 16 days, and the time away from work is correspondingly shorter than is the custom with the more orthodox methods of closure.

Method of Use of Stainless Steel Wire

Two principles must always be adhered to.

First, the anatomical relations of the parts sutured must not be distorted. Distortion implies tension, and tension leads to tissue necrosis. Stainless steel sutures must therefore be laid in the tissues without tension. The dissection prior to suturing or darning need involve only the immediate vicinity of the defect concerned. Fascial planes are thus not disturbed beyond a limited definition of the tissues.

Secondly, the wire must never be kinked. It will break at a kink. Care must therefore be used in all the processes of handling the wire, as well as in its introduction into the tissues.

The Needles.—Ordinary needles can be used, the wire being threaded through the eye and a short length twisted several times on itself. We prefer "atraumatic" needles which are affixed to the end of the wire: "eyeless" half-circle triangular No. 4 for laparotomy incisions and incisional defects, and Koenig's "fish-hook" No. 5 for inguinal and femoral herniae.

The Stitch.—The wire can be used for any type of suture, continuous or interrupted, superficial or deep; but the methods we describe below are those which we have found the most useful.

The first complete stitch is passed through the tissues and the wire tied in a close reef-knot so that the suture is taut but not tight. The short end is then gripped against the knot with a pair of artery forceps, cut level with the blade of the forceps, and the stump twisted 180° into the adjacent tissue. (It is best to keep an old pair of scissors, curved on the flat, specially for cutting wire.) In thin subjects with little subcutaneous tissue it is advisable to cut the loose end of the wire flush with the knot. The wire is then sewn in according to the methods described below, and finished off with a reef knot, the free ends being cut short and twisted in in exactly the same manner as at the beginning.

The assistant, throughout the whole process of sewing, holds the wire in a loose open loop, using both his hands. He allows the wire to follow without tension as the needle is passed through the tissues and catches it again as it is drawn out. By this means kinking is prevented. A convenient length of wire to use is 18 to 24 inches (45–60 cm.).

Closing Abdominal Incisions

Paramedian Incisions.—In our opinion the best method in patients of normal build is that of Babcock. The peritoneum, together with the posterior rectus sheath above the semilunar fold of Douglas, is closed with a continuous catgut stitch in the usual manner. The muscle and anterior sheath are then closed with a running "far-and-near" (i.e., figure-of-eight) stitch. (See Fig. 2.) Each "far" stitch is passed through the anterior rectus sheath and muscle about 1 cm. from the line of section and brought out in the reverse order on the other side, again about 1 cm. from the cut edge. This stitch varies in its exact details, depending on the distance of the incision from the midline, whether it is above or below the semilunar fold of Douglas, and the degree of divarication of the rectus muscles. The "near" part of this "figure-of-eight" stitch takes in only the anterior rectus sheath. Each plane of the wound is thus accurately approximated, but it is most important that the stitches shall be pulled no tighter than just sufficient to draw the cut edges together.

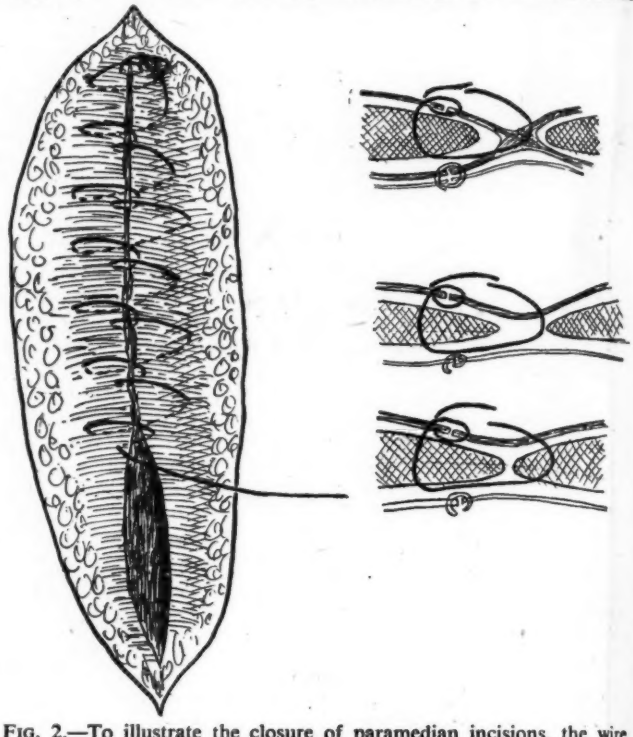

FIG. 2.—To illustrate the closure of paramedian incisions, the wire being retained.

In thin patients it is better to use interrupted "far-and-near" sutures and to cut each wire flush with the knot; or to use the following alternative method, which is convenient when it is required to reopen a paramedian incision. The wire is inserted in a manner somewhat similar to that used in sewing in a subcuticular suture, the anterior rectus sheath being approximated by a flat back-and-forth stitch which is not knotted. (Fig. 3.) The wire is introduced and brought out

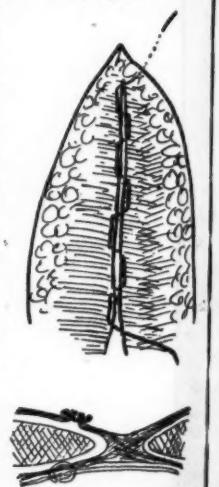

FIG. 3.—To illustrate the closure of paramedian incisions when the wire is to be removed.

through the skin at a little distance from the bottom and top of the incision and the ends are tied together in a loose loop over the inner layer of dressing. After twelve days one end is cut short and the wire pulled out.

Long Oblique or Transverse Incisions.—These are closed by using either a continuous "far-and-near" stitch (similar to that described above for paramedian incisions) or interrupted sutures.

Midline Incisions.—These are closed with a simple running stitch, the bites again being about 1 cm. deep.

The Repair of Hernial Defects

Inguinal Herniae.—Indirect sacs are dissected free, tied off, and removed in the usual manner. Direct sacs are similarly dealt with in cases where they merit such treatment. The transversalis fascia is sutured or plicated if this appears advisable. The posterior wall of the inguinal canal is then reinforced with a steel darn as in Figs. 4A and B. The needle is first passed

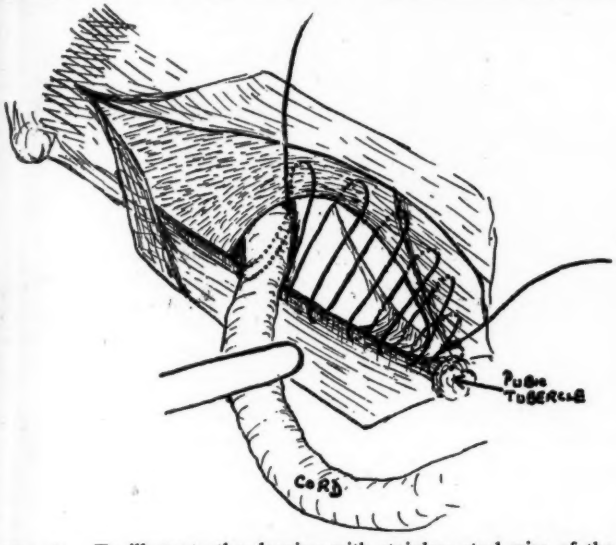

FIG. 4A.—To illustrate the darning with stainless steel wire of the posterior wall of the inguinal canal and reduction in size of the internal ring.

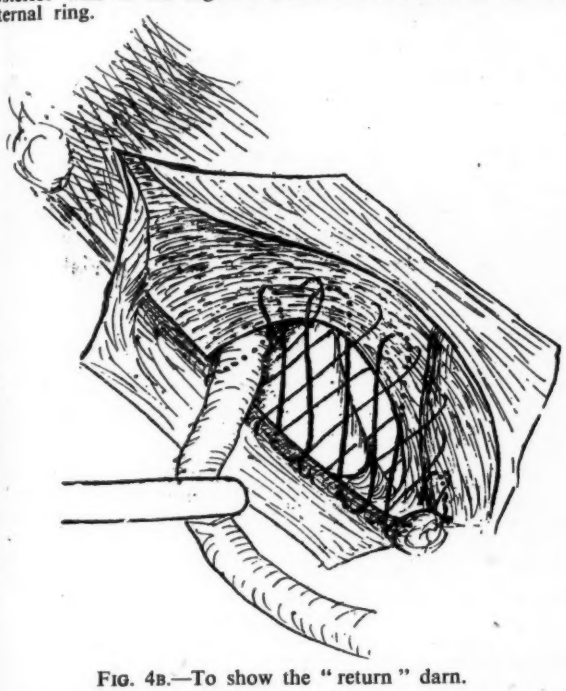

FIG. 4B.—To show the "return" darn.

through the periosteum covering the pubic tubercle, the stitch including the lower end of the anterior rectus sheath and the origin of rectus abdominis muscle. The wire is tied with a reef knot and the short end left about 4 in. (10 cm.) long. The next stitch takes a good bite of the rectus sheath and muscle above, Gimbernat's ligament and inner end of Poupart's ligament below; the next, the edge of rectus and conjoined tendon above, Astley Cooper's ligament and the inner end of the recurved edge of Poupart's below; the next, conjoined tendon above and the recurved edge of Poupart's below; and so on outwards to the internal ring, which is reduced to an appropriate size. Here, if necessary, the wire can be conveniently tied off and a fresh piece used for the return darn, which is brought back in a similar manner to the starting point of the suture. (See Fig. 4B.) This slightly more superficial "return" darn is placed obliquely to the first line of sutures so that a criss-cross or trellis-work pattern is formed. (Fig. 4B.) The upper bites are each at least 1 cm. deep: the weaker the tissues the bigger the bites.

The stitches are put in 5 to 7 mm. apart, considerably closer than in closing abdominal incisions. The wire is laid in the tissues without distorting the anatomical relationships as they are found, and each stitch is gently pressed into place. It is an advantage to have the edges of the divided external oblique aponeurosis held apart by a self-retaining wound-retractor. This tends to fix the gap between conjoined tendon above and Poupart's ligament below, enabling the steel darn to be inserted without drawing the edges of the defect together.

The placing of the stitches requires an exact and gentle touch on the part of the operator and his assistant, who holds the wire in an open loop with both hands as described above.

In some cases, particularly in direct herniae, the transversalis fascia is firmly attached to Poupart's ligament down to the pubic tubercle. Where this is so, it seems better not to break through this barrier in order to include Astley Cooper's ligament in the darn at the inner end, but to use Poupart's ligament throughout as the lower fixation of the wire.

The cord is then placed on the surface of the darn and the external oblique aponeurosis closed with interrupted thread or catgut or steel stitches. The skin is closed with clips.

Femoral Herniae.—These we approach from above. After the sac has been dealt with in the usual manner the femoral canal is occluded with a darn beginning at the pubic tubercle and carried outwards, passing between the outer edge of rectus abdominis and its sheath and the conjoined tendon above and Astley Cooper's, Gimbernat's, and Poupart's ligaments below. (Fig. 5.) The femoral vein is guarded in the usual manner and care is taken that it is not compressed by the outermost stitch. There appears to be no danger of erosion of the vein provided these precautions are taken. The darn is, of course, very much smaller than with inguinal herniae.

FIG. 5.—To show the placing of the wire darn over the femoral canal from above Poupart's ligament.

Recurrent Inguinal and Femoral Herniae.—The technique described above rarely needs to be varied for recurrent herniae, and our methods have so far met with complete success. Size is no contraindication.

Incisional Herniae.—After the sac has been dealt with the defect is occluded with a stainless steel darn which is carried from one edge of the opening to the other, good bites of at least 1 cm. in depth being taken of the tissues forming the ring. It is not necessary to carry out an extensive dissection to define the various layers forming the ring. No attempt is made to draw the edges of the ring together unless they fall into place without tension. For very large defects a length of wire 4 to 5 ft. (1.2 to 1.5 m.) long carrying a needle at both ends is more convenient, and the darn may be done on the "bootlace" pattern. (Fig. 6.)

Normally the darn becomes incorporated in tough fibrous scar so soon that the risk of strands becoming separated and a recurrence developing is remote.

Again, the size of the hernia or its ring is no contraindication to this operation. Any hernia can be dealt with. It is a relatively quick operation. The necessity for extensive dissections around the ring is eliminated. Therefore these repairs can often be carried out in poor-risk patients who would not be

able to stand plastic repairs, Gallie grafts, or operations such as Nuttall's.

Umbilical Herniae.—Where possible we close these by the Mayo technique, using interrupted stainless steel stitches. If there is any tension we use the method described above for incisional herniae.

Hernial repair with steel must be done with great care and precision, but compared with the various fascial graft and skin graft operations the method is simple.

Darning hernial defects with floss silk is easy and effective, but the threat of infection, accidentally introduced at the time of operation or arriving later at the site of the wound by way of the blood stream, caused us to give up this method soon after it had been introduced. There is no need to emphasize the unpleasantness of an infected floss-silk darn.

The stainless-steel wire method is somewhat similar to the silver-filigree operation of McGavin, as practised by Percival P. Cole. The filigree reinforcement, however, is laid over the defect and held in place with small stitches, whereas in the steel-wire method the reinforcement incorporates the margins of the defect in its meshes without strangulation of tissue. Should there be a recurrence following a filigree repair it occurs at the edge of the filigree. The method we describe obviates this source of trouble, in that the reinforcement is fused with the edge of the defect. There is a further advantage that a length of stainless steel wire can be used to darn a hernial defect of any shape or size, whereas filigrees need to be made up in sets and the sets maintained complete.

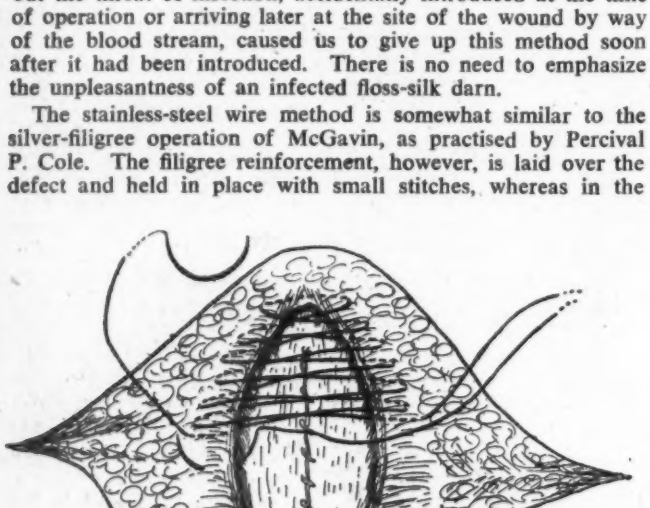

FIG. 6.—To show darning of incisional hernial defect after removal of the sac and closure of the peritoneum. (Needles mounted on each end of long length of wire.)

Some Observations

Wire handles differently from silk, thread, or catgut, but with a little experience it may be used with equal facility. Speed comes with practice, and we now find that closure of abdominal incisions by one or other of the methods described above is quicker than by any other practicable method.

In respect of tensile strength, holding power, absence of inflammatory and foreign-body reaction, rapidity of healing, and in the reduction of complications in an infected wound, stainless steel is better than catgut, linen or cotton thread, or silk. Wounds closed with nylon are more liable to become infected. This is probably due to the fact that it stimulates a fair degree of tissue reaction (Large). Also it is not so strong as stainless steel; it is less secure in the knot, and is more bulky. Tantalum wire induces the same type of favourable response on the part of the tissues and can be used as a monofilament suture. It is easier to use than stainless steel and has less tendency to kink. It is, however, not so strong and more brittle in the knot; it is difficult to procure and very much more expensive.

Dambrin records that exposure to *x* rays is without untoward result, and this has been our experience, though this aspect of the problem needs further investigation.

In about one case in every 200 a broken end of wire causes the patient discomfort and needs to be removed under local anaesthesia. This trouble has not been encountered except in the midline or paramedian closures in thin patients, and it is probably better to use interrupted sutures or the method illustrated in Fig. 3 in such subjects.

The reopening of an abdominal incision closed with wire takes a little longer than when catgut or thread has been used, but the method is easy. The skin and subcutaneous tissues are incised down to the wire loops. These are then defined sufficiently to be pulled up and divided between artery forceps, and the separate segments are then pulled out, care being taken to remove the whole wire. The knife blade used for this part of the incision is rendered useless for further work. There is no difficulty in closing a wound with wire for a second time.

In no case has the wire, or a fragment of it, burrowed into the tissues more deeply and caused trouble.

Summary

In our experience the use of stainless steel wire makes the closure of large abdominal incisions a clean procedure which is completely secure. It eliminates the risk of post-operative disruption and herniation. No such complication has occurred in over 2,500 consecutive major laparotomies, a large number of which were for malignant disease of the alimentary canal. Its use has been accompanied by a considerable reduction in the incidence of infection. Skin scars are neater. It has enabled us with great confidence to practise very early post-operative ambulatory treatment, with the concomitant benefits of a reduced incidence of distension and of pulmonary and vascular complications.

The use of stainless steel darns in the repair of hernial orifices has simplified the operation. Any hernia, however large the defect, can be repaired by this method. The closure seems to be more secure than by any other method in that we have had no recurrence in over 300 cases over a period of nearly five years.

We are of the opinion that the strong, smooth, fine, innocuous filament of stainless steel is at present the best material at our disposal for the closure and reinforcement of fibromuscular planes. It must be used correctly, without tension, without strangulation of the tissues, and without kinking of the wire.

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The Ministry of Health has issued a statement on the remuneration of chemists in the National Health Service. The scheme includes the following details, though interim arrangements will have to be made until they are fully worked out. Ingredients of medicines and appliances will be paid for at current wholesale prices as contained in the N.H.I. Drug Tariff with an additional on-cost allowance of 33⅓% (adjusted as necessary for purchase tax). Dispensing fees will be paid for most prescriptions at an average rate of 1s. each, and containers will be paid for at an average rate of 2½d. each. There will be additional payment for prescriptions marked "urgent" and dispensed when the shop is closed. For organized rota services chemists will receive 7s. an hour on week-days and 12s. 6d. an hour on Sundays and public holidays. Until this scheme comes into operation dispensing fees for prescriptions including those marked "urgent" and made up when the shop is closed will be paid at the current basic rates set out in the Drug Tariff with the addition of 161⅓%.

"MUSHROOM" POISONING DUE TO AMANITA PHALLOIDES

BY

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Edible fungi have been a favourite culinary delicacy for centuries. Mushrooms were recognized as a valuable form of food by the ancient Babylonians and early Romans, who employed them in large quantities not only as a delicacy for the tables of the rich but also as a staple article of diet of the poorer classes.

Although the ancients were possessed of considerable knowledge regarding edible fungi and poisonous varieties were well recognized, many fatalities must have occurred. Records of death from fungus poisoning, often amongst eminent people, figure prominently in contemporary writings, especially those of Pliny; but the earliest record of fatal poisoning dates back over two thousand years. In the fifth century B.C., when Euripides the poet was at Icarus, a woman with her two grown-up sons and a married daughter "were destroyed by pitiless fate in one day" after eating fungi gathered from the fields. In this instance it is probable that *Amanita phalloides* (Death Cap) was the cause of the tragedy; for besides being the most poisonous fungus known it is responsible for more than 90% of all deaths due to mushroom poisoning and is attended by a mortality rate of from 50 to 70% (Ford, 1923). Ramsbottom (1945) states that a death rate as high as 90% has been claimed. Although this percentage is unduly high as an overall figure, there are many examples of a 100% fatality rate in particular groups in which all members partaking of a meal have succumbed (Ford, 1923).

Mushroom poisoning is a commonplace on the European continent, while 200 fatal cases are said to occur annually in Germany alone. In this country but few cases have been reported in the literature. Thus Dubash and Teare (1946), recording four fatal cases, could trace only 21 reported cases of mushroom poisoning with 14 deaths in Great Britain between 1930 and 1942. Birch (1946), quoting from the Registrar General's record, could find only 38 cases of fatal fungus poisoning in England and Wales between 1920 and 1945.

As the time of year is now upon us when *A. phalloides* will again be abundant in the woods and adjoining pastures, and when many continentals are still in our midst, two further cases of Death Cap poisoning seem worthy of report.

The Present Cases

Two German prisoners of war—H. W., aged 29, and J. W., aged 30—were admitted to an R.A.F. hospital on the morning of Sept. 13, 1946, complaining of intermittent seizures of cramp-like abdominal pain accompanied by severe attacks of vomiting and violent purgings. They gave the following story: On the morning of Sept. 11 they had gathered "mushrooms" from a near-by wood; and the next day, after preparation, the fungi were boiled in water for three-quarters of an hour before being fried. The failure of the cooking "mushrooms" to blacken a silver coin was considered proof that the meal would be safe; and each man ate approximately half a pound of mixed fungi. They found the meal most palatable and did not complain of ill effects until nine and twelve hours later respectively. In both cases initial symptoms consisted of severe vomiting, soon to be followed by intermittent attacks of griping upper abdominal pain and diarrhoea. Symptoms continued throughout the night, and on admission to hospital on Sept. 13, eighteen hours after ingestion, diarrhoea and vomiting had taken place fifteen times in H. W. and ten times in J. W.

The patients were first seen personally on Sept. 14, the day after admission. Diarrhoea with vomiting had occurred almost two-hourly in J. W. Fluids by mouth and an intestinal sedative had been prescribed. On questioning the patients the history of ingestion of "mushrooms" followed by a silent period of many hours before the onset of symptoms made a diagnosis of poisoning by *A. phalloides* virtually certain. Treatment was therefore conducted along these lines without waiting for more definite proof. Additional evidence, however, was soon furnished by the patients themselves. One volunteered that the types of fungus they had gathered were chiefly those with large bulbous stems surrounded in their lower parts by a membrane or volva (see Fig.)—characteristics typical of the *Amanita* group. They

Photograph of specimens of *A. phalloides* at different stages of growth. Note the well-marked volva at the base of the stem, the faint dark (green) striations on the surface of the cap, and the dead-white gill-plates seen in the oldest specimen.

also identified a specimen picked by a fellow prisoner of war (who had also partaken of a small quantity of the mushrooms but had been unaffected) as similar to some of the fungi they had eaten. This specimen proved to be *A. phalloides*. Later a personal visit to the wood accompanied by a colleague with sound botanical knowledge showed that in an unselected picking from the area concerned *A. phalloides* constituted about 10% of the fungi collected. *A. rubescens* (Warty Caps), a non-poisonous variety, were plentiful; but no common mushrooms were identified. It was roughly estimated that in half a pound of uncooked fungi each man had probably eaten one or at most two specimens of *A. phalloides*.

Clinical Examination

Seen on Sept. 14, the second day, both patients, of good physique and development, were exhausted and somewhat dehydrated, especially H. W., in whom symptoms had begun earlier and were more severe. Thirst was marked. Mental aberrations or nervous involvement—a muscarine-like effect that is sometimes observed—were not a feature in either case, except that H. W. was a little disorientated and excitable, although able to co-operate and give a rational history. There was no sign of jaundice or haemorrhagic tendency in either case, while lacrimation, sweating, and salivation were absent. The tongue was dry and coated and the breath heavy. The skin and especially the extremities felt cold, but were without lividity or cyanosis; the temperature was subnormal and the pulse small and rapid. On abdominal examination H. W. complained of tenderness under the right costal margin; in neither case was the liver or spleen palpable. No abnormal physical signs were found in the nervous system, and the rest of the examination was essentially negative. The urine, normal to routine tests, was small in amount and concentrated. Vomitus and excreta contained little solid matter, considerable mucus, but no macroscopic blood in the early stages.

Treatment and Management

All efforts to obtain supplies of antiphallinic serum proved unsuccessful. Gastric and high colonic lavage with 1% saline solution was performed three times on the day after admission

and daily for the next three days. On each occasion lavage was continued until the return was clear. This involved as much as three gallons of fluid during the early part of treatment. Two ounces (57 cc.) of 50% magnesium sulphate were left in the stomach. The effect of lavage produced an immediate and striking diminution of vomiting and diarrhoea, which ceased in the case of J. W. after the first treatment.

On Sept. 14, 5% glucose in normal saline, four to five litres in 24 hours, intravenously, was begun; this was continued for four days, together with 15 units of soluble insulin and 10 mg. of vitamin K four-hourly in an attempt to prevent or minimize liver damage, which was fully anticipated. Apart from ice to suck, all fluid requirements were met by parenteral administration. Atropine 1/100 gr. (0.65 mg.) and morphine 1/4 gr. (16 mg.) were prescribed to control restlessness and pain during the first three days.

On the sixth day unlimited glucose drinks and small quantities of citrated milk were substituted for the intravenous drip, but four-hourly insulin injections, combined with vitamin K and vitamin B_1 12,000 units and 10 ml. of 10% solution of calcium gluconate parenterally, were continued for a further forty-eight hours. A light diet rich in protein and carbohydrate was then prescribed and steadily increased until discharge from hospital on Oct. 21, when the patients were taking a normal diet.

Clinical Course of Case 1 (H. W.)

Although gastric and colonic lavage decreased the frequency and severity of vomiting and diarrhoea, both persisted until the evening of the third day, when these symptoms gave way to complaints of upper abdominal pain. At this time the patient was unduly drowsy and lethargic. Examination revealed tenderness under the right costal margin, but the liver was not found to be enlarged. On the fourth day definite jaundice of the sclerotics was noted, but the skin was normal in colour. The urine, small in amount, was dark amber and concentrated. The patient, however, felt much better; drowsiness was less marked and he was more alert; nor was there any complaint of abdominal pain or sign of liver enlargement. Clinical improvement from this point was rapid despite an intermittent pyrexia, which reached 102° F. (38.9° C.) on Sept. 17 and persisted with gradual defervescence until Sept. 27. All signs of jaundice had cleared by Sept. 21, and urinary output, which had previously been unsatisfactory, rapidly improved.

Laboratory Investigations.—Sept. 14:—Urine: N.A.D. *Sept. 17:*—Blood: Hb, 94% (Sahli); R.B.C., 4,280,000; W.B.C., 5,400 per c.mm. (polymorphs 77%, lymphocytes 20%, monocytes 3%). Urine: Urobilinogen +; albumin and sugar, *nil*; no deposit. *Sept. 18:*—Total plasma proteins: 5.5 g. per 100 ml.; albumin–globulin ratio, 1.6:1. *Oct. 2:*—Electrocardiogram: Physiological; no evidence of bundle-branch block (a feature reported by Hyman, 1928). *Oct. 4:*—B.S.R.: 35 mm. in first hour (Westergren). *Oct. 5:*—Total plasma proteins: 6.5 g. per 100 ml.; albumin–globulin ratio, 5:1. *Oct. 7:*—Serum bilirubin: Less than 0.5 mg. per 100 ml. *Oct. 17:*—B.S.R.: 14 mm. in first hour (Westergren).

Clinical Course of Case 2 (J. W.)

On the second day, after the first gastric and colonic lavage, for which three gallons (13.64 litres) were required, the patient felt more comfortable, while vomiting and diarrhoea ceased completely from this time. The patient still complained of pain under the right costal margin, but there was no jaundice or evidence of hepatic enlargement.

The condition appeared to have improved on the third day despite pyrexia to 101.6° F. (38.7° C.). Apart from superficial ulcers of lips and tongue there were no complaints, and the patient felt much better. Clinical examination was negative. Anuria, however, persisted throughout the day, and in the evening his condition rapidly deteriorated. He complained of generalized aching pain of sudden onset and severe right-sided upper abdominal pain, requiring morphine for relief. A bowel action, consisting of a very pale stool, was accompanied by passage of a few ounces of dark amber urine.

On the fourth day, after a noisy restless night when morphine and atropine had to be administered, the patient became more restless and very drowsy, although rousable. His chief complaint was severe abdominal pain. Jaundice of sclerotics and skin, although not deep, was obvious, and pruritus was troublesome. No haemorrhage or petechiae were seen. Abdominal examination revealed extreme tenderness in the epigastrium and under the right costal margin. The liver was enlarged one to two fingerbreadths; the spleen was not palpable. Temperature was 100° F. (37.8° C.); pulse, 64 a minute, and regular.

Restlessness, with sleep for brief periods, occurred throughout the next night; but by the fifth day his condition showed some improvement. Jaundice and hepatic enlargement were unchanged, but abdominal pain and tenderness were less. Examination of the lungs showed coarse basal crepitations, which persisted for several days; there were no respiratory symptoms. Following this acute attack the patient's condition steadily improved, but more slowly than in Case 1. Jaundice, never deep, persisted together with hepatic enlargement until Sept. 28 (16th day), when he was free from all symptoms and taking a light diet.

Laboratory Investigations.—Sept. 17: Urine: S.G. 1020; urobilinogen +; albumin +; sugar, *nil*; deposit—leucocytes with granular debris. Plasma proteins: Total 5.2 g. per 100 ml.; albumin–globulin ratio, 1.8:1. Blood: Hb, 101% (Sahli); R.B.C., 4,820,000; W.B.C., 5,000 per c.mm. (polymorphs 71%, lymphocytes 19%, monocytes 10%). *Sept. 18:*—Serum bilirubin: 1.25 mg. per 100 ml. *Oct. 2:*—Electrocardiogram: Physiological. *Oct. 8:*—Plasma proteins: Total, 6.6 g. per 100 ml.; albumin–globulin ratio, 4:1. Serum bilirubin: 0.5 mg. per 100 ml.

Both patients were confined to bed for three weeks from the time of admission to hospital; they returned to duty on Oct. 21.

Discussion

Amanita phalloides produces two main active principles—phallin and amanita toxin. The former, first described by Kobert (1891), is an alcohol-insoluble haemolysin. It is relatively unimportant in cases of poisoning, for, being an easily decomposed glucoside, it is rapidly destroyed by heat and by digestive juices. Amanita toxin, the essential poison, is an alcohol-soluble indole or aromatic phenol derivative. It is resistant to heat, to drying, and to digestive juices, and in experimental animals it produces most of the lesions which are described in fatal cases of *A. phalloides* poisoning in man (Ford, 1908, 1923).

The susceptibility of different individuals to the amanita toxin varies, but surprisingly small amounts of the fungus and even a portion of a single specimen may prove fatal, especially when eaten raw (Plowright, 1879). The case histories and course of the illness reported above indicate that

Table showing the comparative relationship of early and delayed symptoms and signs

	Period of Gastro-intestinal Symptoms			Period of Delayed Hepato-renal Damage						
Case	Silent Period (Hours)	Severity of D. and V.	Total Length of Symptoms (Hours)	Abdominal Pain and Tenderness	Duration of Jaundice (Days)	Pruritus	Liver Enlargement	Urine	Serum Bilirubin	Total Plasma Proteins. Alb./Glob. Ratio
1	9	+++ 15 attacks before admission	69	+	Slight (6 days)	Nil	Nil	Urobilin. +; albumin, nil; no deposit	—	Sept. 18: 5·5 g.; ratio 1·6 : 1 Oct. 5: 6·2 g.; ratio 5 : 1
2	12	++ 10 attacks before admission	38	+++	+ (13 days)	+	1–2 finger-breadths	Urobilin. +; albumin +; polys. and deposit +	1·25 mg./100 ml.	Sept. 17: 5·2 g.; ratio 1·8 : 1 Oct. 8: 6·6 g.; ratio 4 : 1

only a small quantity of *A. phalloides* had been eaten by the patients—a contingency made all the more probable by the fact that a third individual partaking of the meal was unaffected. Thermostability of amanita toxin was well demonstrated by its resistance to boiling and frying for almost an hour (at a maximum temperature of about 140° C.); the silver-spoon test recommended by Atkinson (1900) proved a misleading and dangerous criterion by which to judge whether the "mushrooms" were safe or not; and, finally, *A. phalloides* was palatable and even pleasant to eat.

The long delay interval of nine and twelve hours respectively before the onset of symptoms emphasizes the important point made by Birch (1946), that in any case of mushroom poisoning an interval of eight hours or more between ingestion and symptoms should be regarded as diagnostic of *A. phalloides* poisoning. The severity of the gastro-intestinal symptoms in the early stages of the illness contrasted with the surprisingly mild hepato-renal damage which took place later (see Table). The importance of eliminating factors in assisting recovery is indicated by the fact that the patient with the shorter silent interval and the more severe and prolonged diarrhoea and vomiting sustained less liver damage than his colleague, whose condition in the early stages was considered the more satisfactory. In addition, poisoning by *A. phalloides* combined with other species of fungus has a better prognosis than poisoning by *A. phalloides* alone, the reason for this being that other types of fungus often cause earlier diarrhoea (Vander Veer and Farley, 1935).

Summary and Conclusions

Mushroom poisoning due to *Amanita phalloides* in two German prisoners of war is described. Both patients recovered.

The severity of the gastro-intestinal symptoms in the early stages of the illness contrasted with the mildness of the delayed hepato-renal damage.

The delayed effects of poisoning were more severe in the patient with the longer silent period before the onset of symptoms and with the less pronounced and protracted diarrhoea and vomiting.

The ability of the gastro-intestinal tract to eliminate the toxin of *A. phalloides*, and the thoroughness with which this elimination is assisted by early and efficient gastric and colonic lavage, are regarded as factors of the greatest importance in determining recovery.

My thanks are due to the Director General of Medical Services for permission to publish this paper and to Professor John McMichael for his helpful criticism and advice. The invaluable assistance of Squadron-Leader Meredith Brown, who identified specimens of fungi, and the keen co-operation of Flight-Lieut. E. R. Arnold, who was responsible for the care of the patients and for carrying out treatment, are much appreciated.

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The Ministry of Health has issued two leaflets explaining how to get dental treatment or sight tests and spectacles under the National Health Service. Members of the public can get copies of them from the offices of the executive councils (address from any post office), at local offices of the Ministry of National Insurance, and at various other centres, such as citizens' advice bureaux.

TREATMENT OF GENU VALGUM

THE DISCARDED IRON

BY

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The psychological effect, both on child and on parent, of wearing irons is usually unpleasant. There are occasionally exceptional people, like the late President of the United States, who achieve superiority in spite of their disability. That is possibly because the essential necessity for wearing irons has been revealed to them and they have been told that irons are necessary for progression or even existence.

Every orthopaedic surgeon who has served during the war must have experienced a thrill on his return to civilian practice on seeing as a young adult apparently cured a child who had been under his treatment five years previously. He may be labouring in the fields; she may be soignée with lipstick and rouge. Yet if treatment has extended over several years it is always easy to detect in these patients a difference from others. An intangible shyness or barrier is present. It might be called an inferiority complex, but it is a sequel of prolonged immobilization, with which social segregation is invariably associated. This result may be regrettable, but it is tempered by the fact that such treatment has been inescapable.

In quite a different category is the child who has to wear an instrument which proclaims to all that he or she is a "cripple," though in truth such an instrument is merely corrective and not essential. Here, leading all the rest, is the knock-knee iron or brace, an appliance of very doubtful value.

This paper is based on impressions derived from the treatment of 8,000 cases of genu valgum treated over a period of 15 years, and has been prompted by seeing children returned to East Anglia after evacuation to other parts of England where treatment was carried out with knock-knee braces and without appreciable results.

Aetiology

Jones and Lovett (1929) describe knock-knee under the heading of rickets. They, however, stress that rickets is not the only cause of knock-knee, and mention tuberculosis of the knee-joint and infantile paralysis as well as fractures in the neighbourhood of the knee-joint with malposition and epiphysial injury. "Loose knees" in children are also mentioned, and I wish to emphasize these as being the chief cause. McMurray (1943) says: "This very common deformity begins as a rule in early childhood, following on rickets," and ten lines later on he states: "As a rule, deformity is due to this overgrowth of the inner side of the femur, the alignment and shape of the tibia remaining normal." Mercer (1943) states: "The deformity of knock-knee develops, as a rule, in early childhood, and is almost invariably due to rickets."

Few orthopaedic surgeons now believe that rickets is the commonest cause of knock-knee, yet this claim holds pride of place in the three textbooks mentioned. I think that knock-knee is due to: (1) laxity of ligaments or loose knees, as mentioned by Jones and Lovett (1929); (2) quadriceps insufficiency; and (3) the child being overweight.

It may be argued that if the knee is loose why does genu varum not occur at the knee instead of genu valgum? The femora are much wider apart at the level of the great trochanters, and slope inwards towards the knee, so that the tendency is normally to valgus. Furthermore, the external lateral ligament of the knee is a thick and very strong structure, reinforced by the ilio-tibial band, and

virtually unstretchable. The internal lateral ligament is a broad thin sheet, and inserted into it is the vastus internus, a muscle which always suffers in any lesion of the knee and, according to Smillie, contracts only in the last 10 to 15 degrees of extension. In a child whose muscles are incapable of supporting the weight-bearing joints adequately there is probably a generalized quadriceps insufficiency, and on clinical examination this is usually most pronounced in the vastus internus, with consequent stretching of the internal lateral ligament. It is much more common to find children overweight since the war, possibly because the diet has contained more carbohydrates—starch without stiffening. Lynx-eyed medical officers of health send to our clinics many children aged 2 with 1 in. (2.5 cm.) of genu valgum. They come so often that it seems that no child aged 2 can be without one inch of genu valgum. This genu valgum is entirely due to laxity of ligaments, and no bony deformity is present as yet.

In my opinion, to say that such a deformity is due to rickets is not true, but when these children are sent to us untreated at the age of 4 or 6 their femora have grown into a valgus deformity because of laxity of the internal lateral ligament and the effect of weight-bearing, and as a result there has been overgrowth of the internal condyle of the femur. I consider that this is the true aetiology and that seldom is rickets the cause.

Diagnosis

The usual method of examining the amount of genu valgum is to measure the intermalleolar distance. This may be a fictitious assessment: (1) because if the angle between the femur and tibia is to remain the same the distance between the malleoli must increase with growth; and (2) one can seldom exert a pressure accurately to equal the weight of the child without causing pain. Therefore not only should the intermalleolar distance be assessed with the child both sitting and standing, but the angle between the femur and the tibia should be noted on each side.

The Knock-knee Brace

Jones and Lovett (1929) devote at least one page to a description of the knock-knee brace; and McMurray (1943), dealing with moulding, says: "As with all rickety deformities, continued moulding is often successful in correcting the knock-knee in the acute stages of the disease, and in a large proportion of children so treated operative correction is unnecessary. If, after moulding, the remaining deformity is slight, a knock-knee iron should be used to maintain and complete the correction. The iron should be removed only when correction has been obtained, or when it is obvious that further improvement is impossible." One may comment that if there is bony deformity moulding cannot help, and if there are loose knees moulding will only make them looser.

Mercer (1943) states that considerable improvement, if not cure, can be obtained by manipulation, and also mentions the Jones walking knock-knee brace.

Treatment

About one patient a year requires operation, certainly not more; wedging of the shoes is carried out in every case, and night splints in the majority. "La vérité c'est dans les nuances," said Voltaire, and this apoththegm can be applied to every branch of orthopaedics or surgery or medicine.

Wedging the Shoes

The shoe is wedged 3/16 in. (0.47 cm.) on the inner side, sole, and heel. When I returned to civilian practice I was impressed by the number of children who had not improved after having only the shoe heels wedged. If the wedge can be incorporated in the upper so much the better. The mothers must be told how important it is that the children wear their wedged shoes all day. A "gramophone record" is quoted, based on a verbal communication from Watson-Jones; and here one cannot stress too much how important it is that the surgeon himself speaks to the mother as follows:

"Your child has knock-knees now. If she is not treated she may grow up with them. Apart from looking unsightly, they may become painful when she grows older. By seeing that her shoes are wedged now you can make sure that she will get better, but she must wear the shoes all the time. That is to say, she must put on her shoes before she gets out of bed; she must walk to her bath in them, and she must not stand up in her bath. If you bring her to see me in six months' time and she has not improved, I will tell you that she has been walking without her wedged shoes."

Cleats or studs may be necessary to keep the wedges constant. As a rule three studs on the outer side of the heel are sufficient.

Night Splints

Night splints produce quicker results than wedging of the shoes alone, but are not prescribed for small amounts of deformity or for fretful children as they may interfere with sleep. Simple gutter splints suffice for small degrees of deformity in young children. In older children with a greater degree we have found the night splints described by Lloyd (1939, 1943) to be excellent, because they do not rotate in the longitudinal axis and therefore remain accurately in the corrective position. Lloyd's night splint (Fig. 1) is based on the mechanics of the Jones knock-knee brace. Fig. 2 shows the knee braced back into full extension by bandaging the limb to the smaller posterior piece. The longer portion of the splint is then locked into position by sliding the tubes A and B over the rods *a* and *b*, and lateral pressure is put on the knee by continuing the bandage round the lateral part of the splint. The bandage is taken well above and below the knee, but the greatest pressure is over the knee area.

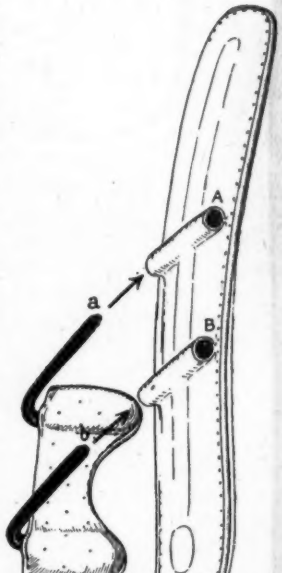

FIG. 1

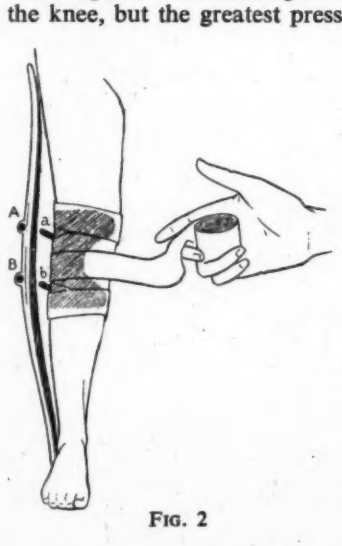

FIG. 2

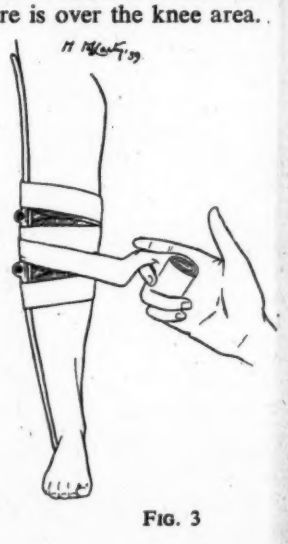
FIG. 3

Osteotomy

An osteotomy is seldom necessary. It is carried out, however, in two circumstances:

(*a*) *For the Child who is Resistant to Treatment.*—These patients are generally over the age of 10, with a neglected deformity and 4 in. (10 cm.) of intermalleolar separation. The osteotomy is performed through a window in a bilateral plaster-of-Paris spica. A line is drawn on the anterior surface of the plaster through the long axis of the limb, an angle being made at the site of the proposed osteotomy. The operation is carried out from the outer side, the plaster first having been cut through two-thirds of its circumference, the inner third remaining intact. The plaster is straightened until the angle no longer exists and the thigh and the shin make a straight line. The plaster is then completed. By this method the possibility of gross displacement of fragments is minimized and a meticulously perfect position is assured.

(*b*) *For Osteo-arthritis in Patients over the Age of 40.*—Knock-knees become arthritic, and this is a point which should be stressed to mothers who are reluctant to have their children's shoes wedged and who consider the application of night splints tiresome and unnecessary. Osteo-arthritis occurs because the outer side of the joint is taking all the weight and the inner side is comparatively idle. In an adult the osteotomy is carried out by linear cuts with an electric saw through the femur just above the knee, and the femur is moulded into the position of correction. I have performed three osteotomies for osteo-arthritis due to excessive knock-knee in patients over the age of 50, with relief of pain and apparently good function.

McMurray suggests an osteotomy through the tibia. Of this I have no experience. Nevertheless I would hesitate to perform a high tibial osteotomy nowadays because of my experience of occasional severe vascular complications after fractures in this region.

The following table gives a working basis for treatment.

2 in. (5 cm.) G.V. below age of 4	Wedge sole and heel only
2 in. G.V. above age of 4 ..	Wedge sole and heel; night splints
More than 2 in. G.V. below age of 4	Wedge sole and heel; night splints
More than 2 in. G.V. above age of 5	Wedge sole and heel; Lloyd's night splints
4 in. (10 cm.) G.V. above age of 7	Osteotomy

This plan may require modification when deformity increases in spite of these conservative measures. That occurs but rarely, and when it does I carry out an osteotomy at an earlier age.

During a recent visit to America I was impressed by the intermediate results of stapling—i.e., the application of staples to control the growth of the lower end of the femur (Blount, 1944). This appears to be a simple, tidy method, less tedious than the prolonged wearing of wedged shoes and night splints, and seems to have a definite place in the treatment of children aged 14–15, in whom shoes have little corrective action and in whom there may be intolerance of night splints. This treatment is, however, *sub judice*.

Conclusions

The psychological effect of knock-knee irons is bad. This is one of the reasons, and an important one, why they should not be used in the treatment of genu valgum.

Genu valgum is seldom caused by rickets. In a large proportion of patients it is due to laxity of ligaments, to quadriceps insufficiency, and to the child being overweight.

Wedged shoes and night splints are sufficient treatment in the majority of cases. Occasionally a supracondylar osteotomy of the femur is desirable in both children and adults.

I wish to thank Mr. Eric I. Lloyd and the Editor of the *British Medical Journal*, by whose permission the illustrations are reproduced. The makers of the splints are Messrs. Beckett and Bird, Bentinck Street, W.1.

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A CASE OF SKIN SENSITIZATION TO STREPTOMYCIN

BY

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The use of streptomycin is restricted at present in this country. The drug is soon to be produced in greater quantities, however, and the time is not long distant when it will become generally available and in common use. It is considered of interest, therefore, to publish details of the following case of sensitization to streptomycin.

Case History

Sister R. W. had had the care of six cases of tuberculous meningitis for four months, during the whole of which time she gave about 40 intramuscular injections of streptomycin a week. The strength of the solution varied between 200 and 500 mg. per ml. and at each injection some soiling of the fingers and hands with the solution was almost unavoidable. At no time did she wear protective rubber gloves.

She was well and did not complain of symptoms during the first three months of exposure, but after this period there was some irritation at night. The little and ring fingers of the right hand and the web between these two fingers were first affected. Two weeks later a similar irritation was noticed on the left hand. At about the same time an eruption suddenly appeared in the flexures of both elbows, and swelling and itching of both eyelids became apparent. There was some rhinitis with a thin watery discharge, and the patient felt generally unwell, but there was no fever.

On examination the contiguous and dorsal surfaces of the little and ring fingers of both hands showed thickening of the skin, with superficial desquamation and fissuring, and there was a slight serous discharge from the lesion. The skin of the web of the affected fingers was reddened, with pin-point papules and a few minute colourless vesicles, some of which had ruptured and discharged a little fluid. A similar fine papular and vesicular eruption affected the flexures of both elbows over an area of 6 by 4 in. (15 by 10 cm.). The skin of the eyelids was oedematous, dusky in colour, and showed a fine desquamation. Nothing further abnormal was discovered on full routine examination.

Patch-testing

Patch tests on the patient and all other members of the nursing staff handling streptomycin were carried out in the following way: 0.2 ml. of 1:5 solution of streptomycin calcium chloride complex (Merck), containing 1 g. of active base in 1.3 g. of substance, was used, drawn from the same batch of drug as is in current use in the treatment of cases in this centre. Sterile water was the solvent. The solution was spread on a single layer of gauze 1 in. (2.5 cm.) square, and the whole was covered with a piece of jaconet, which was held in place with zinc-oxide plaster. A control patch consisting of gauze, jaconet, and plaster was used in each case. The skin was not cleaned as a preliminary. The patch was left in place for 24 hours, and the area was examined at the time of its removal and 24 and 48 hours later.

The patient and seven other nurses who had administered the drug for periods of between one and a half and five months were thus observed. A control group of seven volunteers who had not been in contact with the drug were also tested. In all

cases save that of the patient both the patch and the control were negative. In the patient, at the end of 24 hours an erythematous eruption at the site was apparent, which later became oedematous and itched. The original skin lesions, which had by then almost disappeared following withdrawal from contact with the drug, showed a simultaneous exacerbation. Further patch tests were carried out with solutions of 1:10, 1:50, 1:100, 1:500, and 1:1,000. With the solutions of 1:10 and 1:50 a similar oedematous and erythematous lesion was observed at the site at the end of the examination period, and there was a similar exacerbation of the clinical manifestations in the original skin lesions. A papular erythematous eruption was seen with the weaker solutions, the lesion being minimal in the test to 1:1,000, and no general reaction was experienced. All the control patches were negative.

Discussion

Strauss and Warring (1947) reported four similar cases among 12 nurses in contact with streptomycin. One developed symptoms after a month, whereas the other three required approximately three months' exposure before any changes were noticed. In all cases the eruption first showed upon the fingers, in two the flexures of the elbow were affected, and changes in the eyelids were noted in all four. The clinical development, the pattern of reaction, and the type of the eruption were strikingly similar to that in the present case By patch-testing they detected two further cases of sensitization which had not hitherto shown clinical manifestations. They were able to patch-test their sensitized cases with a solution containing only 2% of impurities, and induced a similar lesion to that produced by a solution with as high a proportion of impurities as was used by ourselves. They conclude, therefore, that the sensitization is probably not due to the presence of impurities. For lack of material it was not possible to confirm their findings.

Crofton and Foreman (1948) described four cases of sensitization to streptomycin, the nurses concerned having been exposed to the drug for periods of between six weeks and six months. Oedema and itching of the eyes occurred in three of them, whilst in two cases the skin of the face was involved. The eruption on the hands and arms was similar to that in the present case. They carried out skin tests, using intradermal injections of 50 μg. of streptomycin hydrochloride dissolved in 0.1 ml. of saline. Positive results were obtained in all four cases, and exacerbation of the original skin lesions occurred in three. The authors failed to detect similar skin sensitization in 30 other nurses who had been exposed to the drug.

The absence of reaction to a 1 : 5 solution in the other exposed personnel and the control volunteers in the present investigation suggests that the solution is not a primary irritant and that this eruption is a sensitization phenomenon. Considering the long period of exposure required before clinical signs become manifest, it seems reasonable to suppose that this sensitization is acquired. Further work is in progress in an attempt to elucidate this point.

Summary

A case of skin sensitization to streptomycin in a nursing sister is reported.

Patch-testing of seven other exposed nurses failed to detect further sensitized cases.

Negative patch-tests in a control group suggest that the drug is unlikely to be a primary irritant.

It is considered probable that the sensitization is acquired.

My thanks are due to members of the Leeds Streptomycin Committee for their helpful advice and their permission to publish this case.

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Medical Memoranda

Gangrene after Scorpion Sting

Scorpion sting causes pain and discomfort of only short duration and serious complications do not usually occur, though slight local necrosis has been seen in some cases. In rare cases, however, especially in children, death has been reported. In the following case gangrene of a finger occurred after a scorpion sting.

Case Report

On Feb. 13, 1947, a young healthy girl of about 17 was stung on the pulp of the right ring-finger by a scorpion which was found and killed. This was followed immediately by the usual symptoms—namely, burning pain, tenderness, and swelling at the site of the sting and cold perspiration of the whole body. A few minutes later a blister appeared just proximal to the point of the sting and it soon spread to and round the middle of the second phalanx of the finger. Potassium permanganate crystals were applied to the point of the sting and a starch poultice to the blister. The latter was applied for about two hours with no effect on the blister, which

burst on the third day, leaving a raw surface underneath. As the tip of the finger was gradually getting dry and black she consulted a doctor on Feb. 22, who advised cleaning the part with spirit and dusting it with "cibazol"—acriflavine powder.

When I first saw her, on March 1, the tip of the finger was dry and black, and proximal to this was a discoloured area which was limited above by a demarcation line. When seen on March 10, the dry black area had extended to the demarcation line (see illustration).

Comment

I could not find any reference to the occurrence of gangrene after scorpion sting in the literature on Indian scorpions. Dr. K. S. Mhaskar, joint author of *Indian Medical Research Memoir on Indian Scorpions* (1932, No. 24), writes in a personal letter that he never came across any case of gangrene after scorpion sting. S. Kubota (1918), however, has reported a case in which local necrosis occurred after a sting by a Manchurian scorpion (*Buthus martensi*). Nowak (1898) showed that when injected subcutaneously scorpion venom, like snake venom, may produce local necrosis.

In the above case the gangrene may have been produced by the action of substances applied locally or by the infection of the part. However, the local application of potassium permanganate to the point of the sting and the application of starch poultice to the blister were not such as to give rise to the production of gangrene in the finger. Infection also does not seem to be the causative factor, as the gangrene appeared to be of the dry type from the beginning. The production of gangrene therefore seems to be due to the action of the venom.

The variety of the scorpion concerned is not known, but it seems probable that the venom of a particular variety produces local necrosis. Though the occurrence of gangrene after scorpion sting might not be very common, the disability it causes should call for further research in this field.

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Reviews

STATISTICAL METHODS

Statistical Methods in Research and Production. With Special Reference to the Chemical Industry. Edited by Owen L. Davies, M.Sc., Ph.D. (Pp. 292. 28s.) London and Edinburgh: Oliver and Boyd.

This volume is the work of seven contributors, and, Lord McGowan states in a foreword, the first of a series of scientific and technical handbooks which Imperial Chemical Industries intend to publish with the aim of making generally available the important information accumulated as a result of the company's manufacturing experience and research. While the company hopes that these publications will be of particular help to industrialists and technicians in industry, this book should certainly be more widely useful than that. Its approach is, naturally, through problems encountered in the chemical industry, and its examples are mainly of the applications of statistical methods in that field. But many research workers with other interests will undoubtedly find it of assistance in learning and applying statistical techniques to any type of data, particularly, it would be fair to add, those who either already have some grounding in statistical theory and methodology or are not perturbed by algebraical symbols. The arithmetician will be aided by the numerous examples that are fully worked out (and upon which—an essential step—he can practise), but much of the discussion and the approach to the arithmetical processes are centred around mathematical expressions.

The book contains chapters on frequency distributions, averages and measures of dispersion, the customary tests of significance, the analysis of variance, regressions and correlation, contingency tables and the chi-squared test, general problems of sampling, control charts, and prediction and specification. A useful glossary of statistical terms is included, and also a list of the statistical and mathematical symbols in general use, with guidance on their type-setting. The usual tables of statistical functions are given at the end—the normal, chi-squared, *t*, variance ratio, binomial and Poisson distributions, the significance levels for the correlation coefficient, and control-chart limits for the average and range. It is clear that the authors have taken great pains over the setting of their text, arithmetical examples, and algebra, and in these austere days it is a pleasure to handle a book so beautifully produced.

A. BRADFORD HILL.

PSYCHO-ANALYSIS AND CHILDREN

The Psychoanalytic Study of the Child. Volume II. 1946. Edited by Anna Freud, Willie Hoffer, M.D., Ph.D., L.R.C.P., Edward Glover, M.D., *et al.* (Pp. 424. 30s.) London: Imago Publishing Co.

It is impossible to do justice to this volume in a short review, so that the reader is advised to get hold of a copy if his interest is in child psychiatry or in psychological theory. Much that is both interesting and clinically important is discussed, and the continued work of the Imago Publishing Company is justified by it. "Problems of Child Development" include a theoretical study of psychic structure, a study of laughter, a further contribution from Mrs. Burlingham on twins, and a study of the pre-oedipal development of the male child. "Clinical Problems" include articles on feeding disturbances by Anna Freud, the psychogeneses of tic and of reading disabilities, the analysis of a child with night terrors, and clinical observation of enuretics, a valuable description of psychosis in childhood by Elizabeth Galeerd, and comments on the diaries of schizophrenics by Willie Hoffer. "Guidance Work" and "Problems of Education and Sociology" are other headings to sections, and there is a historical note on J. B. Felix Descuret (1795–1872).

Only one article can be picked out for detailed comment; that by Rene A. Spitz on what he calls anaclitic depression is chosen, because it involves consideration of Melanie Klein's concept of the depressive position in emotional development. Spitz makes a special point of disclaiming acceptance of this concept. The material of the article is excellent. The infants in an institution who had all been (unavoidably) separated from their mothers at the age of 6–8 months were found to become depressed at 8–11 months. The author gives interesting clinical details.

Klein uses the term depressive position to describe an achievement of the human infant. If all goes well with the emotional development of any infant a stage is reached at which the infant recognizes the results of erotic and aggressive impulses and ideas, so that henceforth he is concerned, is able to feel responsibility and guilt. Only if this stage is achieved is deprivation liable to cause depression or, in the simplest case, mourning. Failure to arrive at, or regression from, this stage indicates a severe (psychotic) disturbance of emotional development, and results in the infant's showing the more primitive defences such as a disintegration of the personality, depersonalization, and loss of contact with reality. But Spitz writes: "Klein assumes that human beings are born with a finished and complete psychic structure." In fact, she does not.

It could be said of the depression observed and described by Spitz that these infants, through early contact with their own mothers and through their subsequent management in the institute, had gone forward in development sufficiently to be capable of becoming depressed, this being an indication of health; more seriously disturbed infants would have failed to achieve this clear depressive mood in reaction to loss. On the basis of an understanding of Klein's theory the author's observation that depression occurred only at 8–10 months is a useful contribution to the discussion of the theory; without such understanding his good clinical observations are wasted. It would be a pity if subsequent volumes must be marred by further uninformed references to Klein. The first volume contained a serious attack on Klein which was too biased to have an effect on scientific thought, but this article loses value through lack of understanding. It is urgently necessary that Klein's critics get to know what she is in fact saying.

D. W. WINNICOTT.

EMERGENCY SURGERY

Emergency Surgery. By Hamilton Bailey, F.R.C.S., F.I.C.S., F.R.S.Ed. Part I. Sixth edition. To be issued in 5 parts. (Pp. 180; illustrated. 21s.) Bristol: John Wright and Sons. 1948.

The sixth edition of Mr. Hamilton Bailey's *Emergency Surgery* is appearing in five parts, of which we have recently received Part I. In size and arrangement it closely resembles one of the six parts of the third and last edition of the same author's highly successful book, *The Surgery of Modern Warfare.* Printing and binding difficulties are apparently still acute, and production in parts to a certain extent overcomes them.

In assessing the qualities necessary for the exacting practice of emergency surgery Mr. Bailey rightly emphasizes physical fitness, mental alertness, and readiness at all times to attend to demands on his services as qualities as important to the surgeon as technical training and experience. However, there is still a need for a guide book, since emergency operations are so often performed by those beginning the practice of surgery. For many years such a guide was to be found in French in the *Traité de Chirurgie d'Urgence* of Félix Lejars, of Paris, who died in 1932, and in the translated English edition of the book by Mr. W. S. Dickie, of Middlesbrough. In more recent times Hamilton Bailey's book has met the same demand, and, now brought up to date in this new edition, it is, like all his well-known publications, an admirable production. The author has already achieved a world-wide reputation as a writer of surgical textbooks which are so well and clearly produced that certain of them have reached the "best seller" class—unusual enough among textbooks to be praise indeed.

In the first volume he discusses infusion and transfusion, shock, restoration of the patient *in extremis* while under anaesthesia, complications of operation such as burst abdomen, and intra-abdominal acute infective lesions such as appendicitis and salpingitis. Among aids to prevent phlebothrombosis he might have mentioned support of the Achilles tendons to keep the calf muscles free from pressure by the operating table, for there is incontrovertible evidence that it is in the calf veins that the condition begins, and if the calves are immobile and lying on the table the vein walls are in apposition and free flow in them is impeded. In a balanced account of drainage, which

opens with the statement that a cynic once condemned books because they do not tell us when to drain, the author would appear to be on the side of the drainers in doubtful cases, but at the same time he well illustrates the harm which drainage tubes may do if used inadvisedly. Among his quotations from various surgeons he might have added the remark of still another surgeon who, when asked why he had not drained the abdomen of a certain important person, remarked, "There was nothing to drain"—sound reasoning enough.

If we may judge the whole from this first part, this edition should be the best yet, and more than ever appreciated by those for whom it has been written by one whose experience in this field is great, and who in the production of this account richly gives of it to his younger and less experienced colleagues.

LAMBERT ROGERS.

UNORTHODOX SYPHILOLOGY

Syphilis. By Henri Mathias. (Pp. 722. 600 francs.) Paris: Librairie Maloine. 1947.

This is a remarkable book; to say that it is unorthodox would be an understatement. After an introduction in which the author points out that syphilis is one of the greatest enemies of the human race, that it can and should be wiped out, and that medical men should be better informed about it, he discusses the subject in five parts: (1) Acquired syphilis, the causal organism, the progress of the disease, its prophylaxis, and its relation to marriage and divorce; (2) treatment, with a criticism of current methods and attempts at shortening the time necessary for radical cure: (3) congenital syphilis; (4) the "patho-genealogical" tree in the uncovering of congenital syphilis; and (5) a consideration of various problems connected with both the acquired and congenital forms.

It is not easy to understand why or for whom this book was written; presumably the author wished to air views which are not generally acceptable. It is not one for the student or general practitioner, though the dilettante experienced syphilologist may be interested and even amused by browsing over the 700-odd pages. It appears that the only remedy for early acquired syphilis is "salvarsan" (the author dismisses penicillin in just over one page) given in a large number of small doses over a period of a year; in cases of congenital syphilis cyanide of mercury and bismuth may be given as well, but must be injected intravenously.

The medical profession in general has no idea of the number of conditions for which congenital syphilis is directly or indirectly responsible; these range from psoriasis to haemophilia and from epilepsy to chronic appendicitis and include most of the ailments due to dysfunction of the ductless glands. The author evidently has the greatest contempt for the average syphilologist for refusing to use the gifts which God and science have given him—eyes to see congenital syphilis all around him and salvarsan. Perhaps the masterpiece of this work is the expression: "Sterilisans magna sed in unum anno" (p. 328).

T. E. OSMOND.

The successive issues of the *Year Book of General Surgery* form milestones by which some estimate may be made of the advances in theory and practice as the years pass; they are also signposts to the general trend of surgical thought and investigation. Thus in the 1947 volume, edited by Dr. Evarts A. Graham (H. K. Lewis; 21s.), we find no less than 55 pages devoted to summaries of articles on the surgery of the blood vessels, and notices of eight papers relating to vagus section in the treatment of peptic ulceration; but pilo-nidal sinuses, which during the war years attracted much attention, are now scantily represented by two articles only. As Dr. Graham says in the introduction, surgical literature has returned largely to a peacetime plane after the large number of articles on military surgery in the preceding years. Work is being done in research laboratories again, and many stimulating articles are appearing. He also makes a graceful reference to our own country: "The British, despite most adverse conditions, are carrying on bravely. This is nothing new for them, as everybody knows. Most of their famous old hospitals were more or less badly damaged by the Nazi bombing, but by the use of make-shifts of various kinds they are continuing to care for patients and to teach students." It is gratifying to read Dr. Graham's opinion of our efforts. As usual we found it impossible to answer more than an insignificant number of the questions asked in the yearly "quiz" on the dust-cover.

BOOKS RECEIVED

[Review is not precluded by notice here of books recently received]

The Stuff We're Made Of. By W. O. Kermack, M.A., D.Sc., LL.D., F.R.S., and P. Eggleton, D.Sc., F.R.S.Ed. 2nd ed. (Pp. 356. 10s. 6d.) London: Arnold. 1948.

An account of biochemistry, including the vitamins and hormones, for the general reader.

Transactions of the Fiftieth Annual Meeting of the American Laryngological, Rhinological and Otological Society. (Pp. 587. No price.) New York: The Society. 1946.

Papers on a variety of E.N.T. topics.

Proteins and Amino Acids in Nutrition. Edited by M. Sahyun, M.A., Ph.D. (Pp. 566. 45s.) London: Chapman and Hall. 1948.

A collection of papers by various writers.

Identification of Tumors. By N. Chandler Foot, M.D. (Pp. 397. 36s.) London: Lippincott. 1948.

A profusely illustrated account of the pathology of neoplasms.

The History of State Medicine in England. By Sir Arthur S. MacNalty, K.C.B., M.A., M.D., F.R.C.P., F.R.C.S. (Pp. 82. 12s. 6d.) London: Royal Institute of Public Health and Hygiene. 1948.

The FitzPatrick Lectures for 1946 and 1947 on State medicine in England from 1837 to 1919.

Disability Evaluation. By E. D. McBride, B.S., M.D., F.A.C.S. 4th ed. (Pp. 667. 72s.) London: Lippincott. 1948.

A textbook of the investigation and evaluation of industrial disabilities.

Treatment by Diet. By C. J. Barborka, B.S., M.S., M.D., D.Sc., F.A.C.P. 5th ed. (Pp. 784. 60s.) London: Lippincott. 1948.

A textbook of diet therapy for the medical practitioner.

The Digestive Tract in Roentgenology. By J. Buckstein, M.D. (Pp. 889. 100s.) London: Lippincott. 1948.

A textbook of *x*-ray examination.

The Childless Marriage. By E. F. Griffith, M.R.C.S., L.R.C.P. (Pp. 206. 8s. 6d.) London: Methuen. 1948.

An account of the causes and treatment of sterility.

Dundee Royal Infirmary. By H. J. C. Gibson. (Pp. 71. No price:) Dundee: Kidd. 1948.

Historical notes on the Infirmary.

Clinical Psychology. By C. Berg, M.D., D.P.M. (Pp. 503. 25s.) London: Allen and Unwin 1948.

The psychoneuroses and their treatment, with illustrative case histories.

Calcium and Phosphorus in Foods and Nutrition. By H. C. Sherman. (Pp. 176. 15s.) London: Geoffrey Cumberlege. 1948.

A general account for students, medical men, and dietitians.

A Manual of Removable Partial Denture Design. By R. J. Godfrey, D.D.S., M.Sc. (Pp. 99. 20s.) London: Geoffrey Cumberlege. 1948.

Intended for senior students of dentistry.

The Reach of the Mind. By J. B. Rhine. (Pp. 188. 10s 6d.) London: Faber. 1948.

A brief account of experimental extra-sensory perception.

Textbook of Anaesthetics. By R. J. Minnitt, M.D., D.A., and J. Gillies, *M.C.*, M.B., Ch.B., F.R.C.S.Ed., D.A. 7th ed. (Pp. 568. 30s.) Edinburgh: Livingstone. 1948.

Much new material has been added to this edition.

Bacterial and Virus Diseases. By H. J. Parish, M.D., F.R.C.P.Ed., D.P.H. (Pp. 168. 7s. 6d.) Edinburgh: Livingstone. 1948.

A short account of the principles of immunology.

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STREPTOMYCIN IN USE

The number of publications reporting results of treatment with streptomycin is now swelling to a flood reminiscent of that produced by penicillin about four years ago. Whereas those from the United States deal with the subject in all its aspects, publications in this country are almost exclusively concerned with the treatment of tuberculosis, our more exiguous supplies having been chiefly devoted to this purpose. Notable among these is the recent report[1] of the Medical Research Council's Streptomycin in Tuberculosis Trials Committee, which decided, on its formation in September, 1946, to restrict the use of the drug to cases of tuberculous meningitis and miliary tuberculosis. The provision of facilities for treating these two forms of the disease has since been greatly extended by the Ministry of Health, and an adequate number of hospitals throughout the country, in addition to the centres operating under the M.R.C. scheme, have been able to obtain streptomycin for this purpose during the past twelve months.

This report deals only with tuberculous meningitis, and is based on 105 cases followed up for a minimum of 4 months; of these patients 71 had died, 7 were stationary or deteriorating, and 27 were doing well. This low proportion of recoveries is unfortunately in accordance with the experience of most others, but some improvement may be expected if earlier diagnosis can be made and in any case when the most satisfactory schedule of treatment has been worked out. The effect of the duration of the disease on prognosis is abundantly clear in this series. Early diagnosis is by no means easy, and an article on the subject by Professor W. S. Craig which appears elsewhere in this issue (p. 374) should be helpful to the family doctor who has not the advantage of laboratory facilities at his elbow. In patients in whom meningitis is accompanied by miliary tuberculosis, retinoscopy may help to confirm an early diagnosis. Professor R. S. Illingworth and Dr. T. Wright describe on page 365 of this issue a study of the occurrence of tubercles of the choroid in 65 patients falling into three categories. Tubercles were observed in 25 out of 42 cases of miliary tuberculosis, with or without meningitis, and changes in their appearance during treatment could be related to prognosis. The importance of age is well seen in the M.R.C. series: in children under 3 the outlook is clearly much worse. It was also shown that intramuscular streptomycin alone is inadequate; intrathecal injections must also be given. Beyond this single fact it seems difficult to draw any conclusions about methods of administration. Among the successful results some patients had long and others quite short courses; treatment during the long courses was in some cases continuous and in others intermittent.

When to stop in an apparently successful case is the most difficult aspect of this problem. Debré and his colleagues, who recently described in the *Journal*[2] a series of 118 cases treated in Paris, continued administration for as long as 10 months. At the other extreme, Glanzmann,[3] of Bern, reports an apparent success after only 15 days' vigorous treatment consisting of intramuscular and intrathecal injections, and he believes that in some cases this may be adequate. It has even been suggested that intramuscular injections are unnecessary. Ketelaer[4] gives a short general account of very favourable results obtained by two groups of Russian workers using intracisternal injections only, 12–20 being given during a period of 2 to 3 weeks. Another valuable contribution came from Oxford, where Sir Hugh Cairns and his colleagues[5] treated 18 cases, of whom 7 have died, 7 are doing well, and 4 are still under treatment. This series is notable for the great variety of surgical procedures used to facilitate both diagnosis and treatment. Ventricular puncture was carried out in all but two cases and streptomycin sometimes administered either by this route or continuously through a tube leading into the interpeduncular space.

At what intervals and for how long to administer streptomycin in treating either meningitis or other forms of tuberculosis is still unsettled. The present tendency is to prolong the interval between doses and somewhat to reduce the dose, but to continue treatment for an arbitrary period of 3 months or more. The best system cannot be rationally worked out until the action of streptomycin is better understood, particularly until it is known whether it has a predominantly bactericidal or bacteriostatic effect. The duration of effective treatment is closely connected with the problem of acquired bacterial resistance. In the M.R.C. series little evidence of this was obtained; 19 out of 22 strains of *Myco. tuberculosis* isolated from the cerebrospinal fluid after a month or more of treatment remained fully sensitive. These tests were made in the Dubos medium, and according to Fisher[6] this may give misleading results. He examined 20 strains isolated from patients after 12 weeks' treatment for pulmonary disease; 5 had acquired resistance and 15 apparently had not, but when these 15 were retested in another medium 6 were shown to be resistant. Subsequent study showed that Tween 80, a constituent of the Dubos medium, may enable a very low concentration to inhibit growth, whereas one a thousand times greater will fail to do so in its absence. Glycerin had a similar though less marked effect. Most studies of the acquisition of resistance have been made in cases of pulmonary disease, in which the repeated isolation of bacilli is relatively simple. It may possibly be misleading to apply these findings to other forms of the disease, but it certainly appears that the efficacy of treatment must often decline

[1] *Lancet*, 1948, **1**, 582.
[2] *British Medical Journal*, 1947, **2**, 897.
[3] *Artzl. Mh.*, 1947, **3**, 877.
[4] *Brux.-méd.*, 1948, **28**, 22.
[5] *Lancet*, 1948, **2**, 627.
[6] *Amer. Rev. Tuberc.*, 1948, **57**, 53.
[7] *Proc. Mayo Clin.*, 1947, **22**, 465.
[8] *J. clin. Invest.*, 1948, **27**, 278.
[9] *Ann. intern. Med.*, 1947, **27**, 989.
[10] Eisele, C. W., and McCullough, N. B., *J. Amer. med. Ass.*, 1947, **135**, 1053.
[11] *J. Lab. clin. Med.*, 1948, **33**, 1.
[12] *Ann. Med. Intern. Fenn.*, 1947, **36**, 575.
[13] *J. Amer. med. Ass.*, 1946, **130**, 485.
[14] *Proc. Soc. exp. Biol. N.Y.*, 1948, **67**, 249.
[15] *Science*, 1948, **107**, 143.

seriously after the first month, and perhaps disappears altogether after 2 or 3 months. Pyle,[7] in the most accurate quantitative study yet made, showed that during treatment bacilli of widely varying degrees of resistance can be obtained from the sputum at any given stage and that a steady shift towards higher degrees of resistance proceeds. After 2 months organisms originally sensitive to about 1 μg. may withstand 100 μg. per ml.; and, after 3, as high a concentration as 1,000 μg. per ml. This change was observed in 5 out of 8 patients studied. Sadusk and Swift[8] report similar findings. Their observations extended to 4 months, and at that stage all strains that could still be isolated showed increased resistance in varying degrees. Whether to continue a policy of long courses of treatment in view of such findings is a matter for serious consideration.

Perhaps the greatest unsatisfied popular demand for streptomycin is for pulmonary tuberculosis. The profession can do much to allay anxiety and even indignation about non-availability of the drug for the average patient by acquainting the public with the fact that for the great majority it would be of little benefit. Recent reports from the U.S.A., such as Muschenheim's,[9] confirm earlier findings that chronic lesions with fibrosis and cavitation respond comparatively poorly, though chemotherapy may help by making thoracoplasty practicable when previously it was not. Apart from cases of miliary tuberculosis, streptomycin is chiefly indicated for early acute exudative lesions; laryngeal and tracheo-bronchial ulceration also respond favourably.

Another and even larger field for the use of streptomycin is in the treatment of infections of almost all kinds due to many species of Gram-negative bacilli. Most of these are highly resistant to penicillin and almost all are sensitive to streptomycin *in vitro*. It does not follow that the infections caused by them will respond to treatment; typhoid fever illustrates this, and undulant fever can be arrested only if full doses of sulphadiazine are given in addition.[10] Bacillary dysentery has been successfully treated: so also, according to Pulaski and S. F. Seeley[11] and Leisti[12] has acute gastro-enteritis of unknown aetiology in infants. This may be an important use, for the drug can be given by mouth for such conditions, being neither destroyed in nor absorbed from the alimentary tract. Streptomycin is indicated in tularaemia and plague, also in meningitis due to any Gram-negative bacillus, notably *H. influenzae*, any Gram-negative bacillus septicaemia, and Friedländer's bacillus pneumonia. Large quantities of the drug have been used in the U.S.A. for treating urinary tract infections, with a fairly constant proportion—50% or more—of complete successes. The causes of failure are not perfectly understood, but they include obstructive lesions and probably incomplete alkalinization of the urine before treatment. Whatever the cause of failure, it is accompanied by the acquisition of a high degree of resistance by the infecting organism, and second courses are therefore fruitless. Some bacteria not only become completely resistant to streptomycin but actually dependent on it for growth. This was first shown with meningococci by Miller and Bohnhoff,[13] as noted in these columns some months ago. The same extraordinary change has now been observed by Rake[14] in *Bact. coli*, and by Paine and Finland[15] not only in *Bact. coli* but in *Staph. aureus*, *Ps. pyocyanea*, and *Proteus morgani*. These authors conclude on evidence based on this change that streptomycin probably acts as a metabolite antagonist. Whatever its precise significance, this sort of behaviour bodes ill for the ultimate future of streptomycin treatment. Acquired resistance is a permanent character, and it may not be long before such strains, derived from a case in which treatment failed, begin to be found in previously untreated patients.

B.M.A. CRITICIZED

When such a vast scheme of reform as the National Health Service Act is introduced the transition from the old to the new is unlikely to be smooth. For six years the British Medical Association, through its Council and Representative Body, argued the case for the medical profession with three successive Ministers of Health, and during this period was subjected to criticism, some of it constructive and much of it destructive, for the way it conducted its business. It has been a difficult task for a voluntary organization to represent the interests of more than 50,000 medical men and women who by the nature of their work are self-reliant individualists. The onlooker who thinks he sees most of the game is at times tempted to think he can play it better than some of those trained to do so. No organization would claim to be perfect or free from the human capacity to err. But so long as it has put its hand to its task in good faith and with courage it can withstand the criticisms levelled at it. The correspondence columns of this *Journal* provide an open forum for medical men and women to discuss freely medical and professional problems, and to be outspoken in their criticism of medical institutions and persons in responsible positions. The catharsis of open criticism has its benefits.

In our correspondence columns this week Dr. W. A. Bourne once more shows himself to be a somewhat severe critic of the Association to which he belongs, and in particular blames it for having concentrated on general principles to the neglect of the details of terms and conditions of service. But what would have been the position if the B.M.A. had entered into detailed discussions on the terms and conditions of service before the major controversies had been settled? There was, indeed, widespread anxiety in the profession lest the B.M.A. should, by negotiating on terms and conditions of service, thereby unwittingly prejudge the issue of whether or not to enter the Service. Many were ready, for example, to criticize the B.M.A. for discussing the question and amount of compensation in relation to the prohibition in the Act of the sale and purchase of practices. In the face of a great deal of opposition among its members, the B.M.A. nevertheless had the wisdom to do this, and also, through the Spens Committee, to discuss in some detail the remuneration of general practitioners. The recommendations of the Spens Committee were accepted by the Government and welcomed by the majority of general practitioners. There would seem, therefore, to be little that is substantial in Dr. Bourne's criticism beyond the sincerity with which he sustains it. And when he deplores the present situation "in which

regulations are pouring out from overworked Civil Servants who know nothing of practical detail," why does he not fix the blame for this on the Minister of Health, who refused to postpone the appointed day so that these matters could be considered more carefully once the profession had decided to co-operate with the Government in launching the new health service? The obsessional hurry of the Government to legislate is the cause of the sense of frustration Dr. Bourne diagnoses as prevailing among medical men and women at the present time. Yet in spite of this the National Health Service has started off much more smoothly than anyone expected, and surely Dr. Bourne is wearing blinkers when he suggests that the B.M.A. should have had "plans ready in advance of the Ministry." He seems to have overlooked the fact that the B.M.A. neither framed nor is operating the National Health Service Act. Dr. Bourne continues to wield his *malleus maleficarum* by condemning the Association's proposals for the representation of the interests of consultants and specialists, and ignores the recent move made by the B.M.A. in conjunction with the Royal Colleges and the Scottish Royal Corporations. These things are now being hammered out, and destructive criticism will be useful only in so far as it prepares the ground for constructive proposals.

That there should be unrest at this moment is, as has been stated, to be expected. Critical voices were heard at the Annual Representative Meeting at Cambridge, and have been echoed in letters to the *Journal*. The Winchester Division of the B.M.A. has recently circulated a memorandum on the organization of the B.M.A. in which it is interesting to find the exact opposite of Dr. Bourne's criticism. "Members have been dismayed," the memorandum states, "to find that while they have been engaged in the battle of general principles—capitation fee versus salary, etc.—negotiations of which we were not fully informed appear to have been proceeding over terms and conditions of service. . . ." This underlines what has been stated above—namely, that members of the B.M.A. generally would have disapproved of negotiations over terms and conditions of service during the controversy on general principles. The Winchester memorandum criticizes the method of election to the B.M.A. Council, the Representative Meetings ("These are a shambles—too many members discussing too many resolutions"), and the Headquarters staff for "being out of touch with the rank-and-file of the profession," although the extension of the work of the staff in the Divisions which the Council has lately arranged provides greatly increased opportunities for the periphery to make its views known at the centre. The fact that such criticisms are made is a healthy sign of an active interest by members in the welfare of the Association. It is good that they should come out into the open, for criticism that is stifled breeds resentment. With the introduction of the National Health Service the B.M.A. will pass into a new phase of its existence, and its organization will continue to evolve to meet the needs of its members. It may be hoped that if the debate continues room will be found for constructive suggestions. Some are made in the memorandum of the Winchester Division and will, no doubt, be considered when the holiday season is over and the newly elected Council of the Association begins its year of work.

"MUSHROOM" POISONING

About two hundred varieties of edible fungi grow in England, but most of us would not risk eating any except the usual variety. The term "mushroom" is often used to cover all varieties of edible fungi, but it is preferable to limit it to the field or cultivated mushroom (*Psalliota campestris*). Though many edible fungi are peculiarly shaped and coloured, the most deadly of the poisonous ones, *Amanita phalloides* or the Death Cap, is easily mistaken for a mushroom. It grows mostly in woods, however, where mushrooms are rare, and its gills are permanently white, whereas those of the mushroom never are. The popular tests of edibility are all fallacious, and safety lies in being able to recognize the Death Cap on sight (chiefly by its white gills) or in making sure that only undoubted mushrooms are eaten. The Death Cap must be avoided at all costs, because poisoning by it is usually fatal. Krause[1] reported 7 deaths in 8 cases, and several similarly dismal series have been described.

The two cases of Death Cap poisoning described elsewhere in this issue by Dr. D. Lewes illustrate several characteristic features, including the fact that prolonged cooking does not destroy Amanita toxin. Both patients recovered without specific treatment, possibly because the poisonous fungi were well diluted with edible ones. Certain other fungi such as *Amanita muscaria* and *Inocybe fastigiata* contain muscarine, which causes blurred vision, sweating, and giddiness soon after ingestion. Three such cases were described in this journal last year by Wilson.[2] Mild effects resemble alcoholic intoxication, and certain Siberian tribes eat fungi for this reason.

From the point of view of treatment, cases of "mushroom" poisoning fall into two groups according to whether the symptoms are immediate or delayed. Those with early symptoms should be given atropine by injection and magnesium trisilicate or charcoal by mouth after gastric lavage. A delay of 8 to 12 hours in the onset of symptoms is characteristic of Death Cap poisoning, and this in itself, as urged by Birch,[3,4] justifies prompt specific treatment. In addition to gastric lavage and measures to prevent failure of liver function anti-phallinic serum should be used if obtainable in time (a limited supply is kept at the Central Public Health Laboratory, London, N.W.9, Tel. No. Colindale 6041). Meusel and Orzechowski[5] recommend the use of choline chloride by intravenous drip and report two recoveries on this treatment.

The rabbit stomach–brain treatment of Limousin and Petit[6] must be mentioned because it was based on experimental observations and has been used with success. Unlike cats, rabbits do not die after eating the Death Cap; but the juice of the fungus is fatal if injected into rabbits, suggesting that the rabbit's stomach can destroy the toxin. Cats fed on *Amanita phalloides* mixed with rabbit's stomach survive several days, and if rabbit's brain is added they recover completely. However impracticable this treatment may seem in a very ill patient, the desperate nature of the emergency merits its consideration. The procedure is to give five uncooked minced rabbits' brains and stomachs daily for several days, but the treatment will be useless if started late. Melendro[7] found it impracticable. Eight of his nine patients died—the survivor being a woman five months pregnant.

Proof that *Amanita phalloides* is the cause of symptoms should be sought by inspecting any fungi left over from the meal and by having the vomit or stomach washings examined

[1] *Med. Klinik.*, 1947, **42**, 458.
[2] *British Medical Journal*, 1947, **2**, 297.
[3] *Practitioner*, 1946, **157**, 135.
[4] *British Medical Journal*, 1947, **2**, 348.
[5] *Ärzt. Wschr.*, 1947, **1/2**, 961.
[6] *Bull. Acad. Méd., Paris*, 1932, **107**, 698.
[7] *Rev. clin. esp.*, 1945, **17**, 211.

for the characteristic spores. Because a meal of "mushrooms" may contain more than one type of poisonous fungus it would be wise to do this even in cases with early symptoms because of the possibility of the late onset of Death Cap poisoning.

SKIN SENSITIVITY TO STREPTOMYCIN

A number of side effects have been reported from the use of streptomycin parenterally, including fever, headaches, pain at the site of injection, vestibular and eighth nerve injury, joint pains, and drug rashes. Pulaski and de Bakey[1] found that drug rashes occurred in 5% of their cases; they included urticarial, erythematous, maculo-papular, and haemorrhagic rashes. It would seem that antibiotics derived from fungi like penicillin and streptomycin are particularly apt to give rise to eczematous rashes from external contact. Strauss and Warring[2] reported a series of six cases occurring in twelve nurses administering streptomycin and rinsing out syringes. Patch-test investigation confirmed the diagnosis. Recently Crofton and Foreman[3] described a similar occurrence, and Dr. C. Stringfellow reports a further case in this issue of the *Journal* (p. 387). The wearing of rubber gloves and avoidance of direct contact are suggested as precautionary measures.

THE DANGERS OF TAR

Tar and its derivatives have long been notorious as a cause of skin diseases in those who work with these substances. Even the fathers of industrial medicine were aware of the malignant sores which attacked men who worked in pitch. Tar also has the sinister property of being the source of the anthracene oils—the principal high-temperature constituents—which are the only definite chemical compounds isolated up to the present time known to be actively carcinogenic. Of these the best known is benzopyrene 3, 4, which is present in coal tar to the extent of 0.003%. Others which can be derived from anthracene but which perhaps are not actually present in tar are dibenzanthracene and methylcholanthrene. Among the great numbers of men employed in handling tar and its derivatives fortunately only a very small percentage develop cancer of the skin, though a great many more suffer from milder conditions, some of which can be considered precancerous. These have been the subject of study by many writers, and the latest among these is Dr. Philip Ross, whose interesting paper appears elsewhere in this issue (p. 369). He finds that tar workers are prone to develop a rather peculiar erythema which is brought on by sunlight or strong winds. Other conditions are acne, tar melanosis, and also a characteristic change in the skin which was christened by Prosser White "shagreen" skin and is marked by keratosis, pigmentation, and telangiectasis, with superficial fissuring and the possible development of papillomata. It seems to take about ten years of working in tar before shagreen skin develops, and there are very few subjective symptoms. Curiously enough, workers in tar seldom contract allergic eczematous dermatitis, the type of "dermatitis due to dust or liquids" so familiar to those who until the "appointed day" assisted in the administration of the Workmen's Compensation Act of 1925. This is the more remarkable as many derivatives of tar are well known for their harmful effects on the skin.

The new growths which may be caused by exposure to tar range from simple keratoses through papillomata of various kinds (which, however, should all be distrusted as precancerous) to epitheliomata. Great variations are reported in the length of time required to produce the latter in tar workers. Ross states that it may be anything from 18 months to 34 years. He discredits, however, the dramatic case first reported by Bang of an epithelioma which resulted from a single splash of tar, a case which made so much impression that it has since been quoted repeatedly. The cessation of handling of carcinogenic substances by no means insures immunity, since growths have appeared after a latent period of many years. Fortunately tar epithelioma is a growth of low malignancy, and Ross recommends the employment of the contact low-voltage *x*-ray apparatus as the treatment of choice.

Since tar cancer was first made notifiable in 1920 there has been a steady increase in the yearly notifications. It is, however, probable that this increase is more apparent than real and is due to the increasing vigilance and interest of officials and medical officers. Prophylaxis is of course most important, and Ross suggests that this problem should be tackled from several directions. So far as possible all processes involving tar and pitch should be mechanized, and actual handling should be avoided wherever possible. Fumes and dust should be removed through efficient exhausts. Protective clothing of stout material should be provided, and workers should be trained to work as cleanly as possible and to change their underclothing daily, or as often as the coupon situation permits. Above all the use of "any old suit," thoroughly impregnated with tarry residue, is to be avoided. On the more positive side the employment of barrier creams is a distinct help—Ross mentions that Professor Kennaway has found that the use of a protective cream on the skin of a mouse considerably delayed the appearance of experimental tar growths. Although many more cases have been notified of late years the number of deaths from tar epithelioma has diminished.

VOLUNTARY ACCELERATION OF THE HEART

Emotional individuals often have a persistently rapid pulse. Sometimes the tachycardia may reach 140 per minute, and such cases present obscure problems similar to those of effort syndrome. It is difficult to imagine how processes in the cerebral cortex bring about such a puzzling state of affairs. There are, however, normal healthy people who can accelerate the heart at will. In most of the recorded cases the possessors of the attribute have been medical students or physicians, and it is quite possible that this exceptional type of response may be much commoner than is generally imagined. Feil[1] and his colleagues have recently reported the case of a medical student who first observed his ability to accelerate his heart while recollecting a terrifying nightmare. During the period of acceleration he developed paraesthesiae in the hands and feet, tinnitus, and palpitation. He could stop the acceleration at will, whereupon the symptoms disappeared and he was left only with a feeling of fatigue. Quite apart from this ability, however, he knew that he was liable to attacks of tachycardia which he could not control voluntarily. When an electrocardiogram was taken it was found that he also had the Wolff–Parkinson–White syndrome of short PR interval and a bundle-branch-block type of ventricular complex. When he was given atropine he was able at first to accelerate his heart beyond the intermediate rise produced by the drug, but with full doses no further acceleration was possible. This study shows that a relationship exists between cerebral activity and the autonomic control of the heart rate. The voluntary acceleration of the heart in the individual concerned may have been made easier by his liability to attacks of tachycardia as part of the Wolff–Parkinson–White syndrome.

1 *Surgery*, 1946, **20**, 749.
2 *J. invest. Derm.*, 1947, **9**, 99.
3 *British Medical Journal*, 1948, **2**, 71.

Amer. Heart J., 1947, **34**, 334.

INTERNATIONAL CONGRESS ON MENTAL HEALTH

GREAT ASSEMBLIES IN LONDON

An International Congress on Mental Health, attended by 2,000 delegates from more than fifty countries, was held at the Central Hall, Westminster, from Aug. 11 to 21. The sessions on the first four mornings were under the auspices of the International Committee on Child Psychiatry, and on the first four afternoons under those of the International Federation for Medical Psychotherapy. During the second week the arrangements were made by the International Committee for Mental Hygiene, and the discussions then took a wider and less technical field.

The President of the Congress, Dr. J. R. Rees, opened it with a speech of welcome and read a number of messages, including one from the World Health Organization and another from Mrs. Eleanor Roosevelt. The Congress was under the patronage of the Prime Minister and Mr. Eden, and its officers and committees included almost everyone prominently associated with psychiatry and psychology and mental health in this country.

In the second week of the Congress, in addition to the plenary sessions, over twenty organizations held specialist meetings. Speeches were in English and French, English speeches being simultaneously heard in French by means of an arrangement of earphones, and French speeches interpreted into English subsequent to delivery. The arrangements for the Congress, which have necessitated more than a year's hard work, reflected the greatest credit upon the organizing committee under the chairmanship of Dr. Rees, the Congress organizer, Mr. Michael Harvard, and the chief administrative officer, Miss Judith Jackson. Social events in connexion with the Congress included a Government reception, and receptions given by the Royal Society of Medicine, the British Medical Association, and the London County Council.

First Day

AGGRESSION IN RELATION TO EMOTIONAL DEVELOPMENT

The first session was devoted to the subject of aggression, which was introduced by Dr. FREDERICK ALLEN (Philadelphia). He concentrated on the positive aspects of aggression—the "going out" quality, as he called it. Aggression, he said, had become a sinister word, and with adequate justification, both in individual and in group behaviour. But it was necessary to restore to the concept of aggression the value of the "going out" quality in all individuals—and nations for that matter—as a condition of creative and responsible action. "We want more of our children to develop the capacity to 'aggress and find their place,' not just as faithful precipitates of social forces, but as creators of the quality needed to sustain the virility of the race."

Dr. NELLY TIBOUT, director of the Child Guidance Clinic, Amsterdam, described experiences with Dutch children during and since the occupation. One of the few clinical pictures practically unobserved before the war was of children who presented an almost complete loss of connexion with reality, while in a clownish way making superficial contacts in an attempt at recovery. Tremendous fears of being able to destroy and of being destroyed played a part in the mental life of very young children. The alarming fact had to be faced that many children in different countries had been placed in a situation which caused them to live on the borderland not only of physical but of psychical starvation.

Miss ANNA FREUD, who was received with special acclamation, described the Freudian theory of aggression in which the aggressive urges were considered to be the representatives of one of the two fundamental instinctive forces—that is, of the destructive instinct or death instinct. Their counterparts were the sex urges as representatives of the life instinct. Normally the development of aggression was intimately bound up with the developmental phases of infantile sexuality. Without this admixture of aggression none of the sex impulses could ever reach their aim. On the other hand, through this constant fusion with erotic impulses the aggressive urges were deprived of their destructive qualities and utilized for the purposes of life.

The general discussion which followed was scrappy owing to the severe time limit which had to be imposed on speakers. Dr. JOACHIM FLESCHER (Italy) said that the fact was usually overlooked that the aggressive instinct, though primary, hid as a rule in the form of an alloy with its counterpart, the erotic instinct. Under the impact of frustration, especially in the autoerotic stage, there was not only danger of aggression but of what he called "pathological progression." Dr. MELANIE KLEIN said that during the first three or four months of life destructive impulses, persecutory anxiety, and splitting processes were at their height. In states of anger and hatred, aggression and persecutory anxiety reinforced each other. In normal development at about 4 to 6 months of age, when love and hatred—libido and aggression—towards the mother came closer in the infant's mind, his anxiety lest she be destroyed as an internal and external object by his sadistic impulses and fantasies led to depressive feelings, a sense of guilt, and the urge to make reparation. If such anxieties in early infancy were excessive in relation to the capacity of the ego to deal step by step with them, the result might be the pathological development of the child.

After contributions from Polish, Greek, and other delegates, the discussion was summed up by Dr. EMANUEL MILLER (Maudsley Hospital), who referred to the growth of the aggressive process as both offensive and defensive. It represented a quasi-attack upon environment but also the creation of a defensive ambience, very much as certain powers surrounded themselves with buffer States. In inquiring into the life history of the child it was his habit always to include not only the genetic factor in the family but also intrauterine activity during the last period of gestation. He found that children active in the uterus during the last few weeks of gestation tended to be the active, forthcoming, and aggressive children.

GENESIS OF GUILT

This subject at the second session was opened in three papers, by Dr. VAN DER WALLS (Amsterdam), who expounded the psycho-analytical theory of guilt, Dr. A. HESNARD (Toulon), who spoke as a psychiatrist, and the Rev. T. GILBY (Cambridge), who presented the religious viewpoint. "Probably," said Mr. Gilby, "the maddest people are not seen by the medical psychologist, just as the most vicious people are not seen by the priest." The discussion was continued by delegates from Copenhagen and Harvard and by two French psychiatrists.

Dr. ERNEST JONES, who presided, summed up the discussion by saying that it was a fundamental question, both philosophical and sociological, whether the sense of guilt was primarily inborn or acquired. The religious view was that it was inborn, and the Christian view that it dated from the fall of man. The decision on the question whether it was innate or not would determine whether a sense of guilt was normal or not. One view was that the individual began with a norm or conscience and experienced morbid deviations from it. Freud put forward the view that guilt proceeded from parental condemnation and punishment; this, being absorbed into the individual, produced a sense of guilt. The sense of guilt did not develop along a straight line; it interacted with other emotional attitudes. Fear, for example, might lead to a sense of guilt, and guilt equally to a sense of fear. "Conscience doth make cowards of us all." Similarly hate could generate guilt, and guilt could generate hate. Thus there were a series of vicious cycles which obscured the tracing of the simple instinct. The complexity of this problem called for much further investigation.

Second Day

THE FAMILY BATTLE-GROUND

Another aspect of aggression, this time in relation to family life, was discussed on the morning of Aug. 12. The chair was taken by Dr. GEORGE HEUYER (Paris), who said that the definition of aggression was still awaited, but whatever the definition one essential element was the tendency of the subject to attack antagonistic individuals or groups. The most important aspect of the matter was the relation of the child to other members of the family.

Professor VITOR FONTES (Lisbon) spoke of the forms of aggression before and after puberty. At puberty the psycho-

physical factor that led mostly to aggressive reaction was sexuality, which at this stage of development was strongly defined. It was thus that in the transferred forms of aggression there appeared occasionally sexual perversions with a more or less apparent sadistic content. The family, in spite of certain inconveniences, was the best organization for the affective education of the child.

Dr. TORSTEN RAMER (Stockholm) said that the most important prophylactic measure against aggression was a psychological understanding by the parent of the individuality of the child and his reactions. Each child should be handled according to his peculiarities and his stage of development. Not only parent but pre-parent education was important. Certain social measures were also to be recommended. The work of mothers outside the home should be limited; at all events it should be no more than half-time. Cramped housing favoured the occurrence of aggression in the family, as also, according to Stockholm experience, did the crowding of large families into blocks of dwellings in the same area. Collective child welfare also led to an increase of aggressiveness if the groups were too large.

Professor D. R. MACCALMAN (Aberdeen) agreed in magnifying the home, which made possible an adjustment therapy for the child. Aggressive tendencies could best be transmuted against a background of stable affection. If aggression was not thus absorbed and controlled in early life, any later attempt would prove difficult and costly. His own experience had convinced him that the early stages of development would be less productive of aggression if a determined attempt were made to give parents some knowledge and understanding of the fundamental needs of young children. As matters stood, parents took this fundamentally important task without guidance. Even the worst of bad parents met with in a child-guidance clinic responded to careful influence. "There is much evidence to show that the bad parent is better than none at all." In a wider application of the subject Professor MacCalman quoted a Chinese proverb: "If there is righteousness in the heart there will be beauty in the character; if beauty in the character, harmony in the home; if harmony in the home, order in the nation; if order in the nation, peace in the world."

The general discussion was opened by Dr. RENE DE MONCHY (Sweden), who said that the existence of aggression in family life should not be regarded as an unfortunate occurrence but as a necessity. It was usual to associate the word with the externalized form of aggression, but the aggression directed inwards was of equal importance. Sometimes it took the form of masochism, but that was only the extreme and pathological form of a normal and necessary impulse. He instanced the manner in which parents repeated in their attitude to their children their own childish experience. A young mother, when asked by her hostess whether her little son would like some cake, replied immediately, "Don't give him anything," and afterwards realized that she was literally repeating what her own mother used to say when out visiting, and how much she had resented it. Professor F. K. GOKAY (Istanbul) gave an account of social work in Turkey, where they are investigating the effect of gangster films in manufacturing adolescent criminals, and also where they have prohibited accounts of suicide in the press. Dr. L. CH'ENG (China) said that psychiatry was very new in China, and there were few psychiatrists in that country. Family relationships had helped to maintain stability in Chinese society for generations, but under the impact of western civilization standards were breaking down. However, there were very few cases of senile psychosis in China, thanks to the fact that old people did not worry about what was to become of them, knowing that their children would look after them.

The discussion was continued by Dr. KATE FRIEDLANDER (London), Dr. KAREN SIMONSEN (Denmark), and Dr. ERNST KRIS (New York), who warned against too sharp a division as between the loving and the rejecting mother. Even the most loving mother was not loving to the same extent at all times, and not all the needs of the child would find in her equal facilities for fulfilment. Professor MACCALMAN, taking up the contribution of Dr. Ch'eng, said that chess originated in China and seemed to him an excellent sublimation of aggression in war. Indeed, it was traditional in this country that sport canalized aggressiveness, and the time might come when the playing fields of Eton would be significant of a greater achievement than the winning of the battle of Waterloo.

DYNAMICS OF PSYCHOLOGICAL DISORDER

At the afternoon session, under the chairmanship of Professor H. FULCHIGNONI (Rome), the Congress returned to the subject of guilt, and, as on the previous day, the religious and the psycho-analytical conceptions were contrasted. Professor D. BRINKMANN (Zurich) read a highly philosophical discourse on the subject, and he was followed by Dr. JOHN RICKMAN (president of the British Psycho-analytical Society) with a paper emphasizing the depressive aspects of guilt. Dr. Rickman said that guilt could not be considered in isolation. It was one kind of psychical action in a complicated, interconnected set of psychical actions which appeared to serve the purpose of keeping the organism in a quasi-stable equilibrium. He listed a number of reactions to guilt, such as the drive to restitution, the compulsive urge to propitiation of an aggressor, and an undue amount of social conformity, compulsive behaviour, and so on, and the wide range of cases of sexual anomaly in which the sexual act was felt to be an injury to the partner or at least a degradation (leading to impotence or frigidity or to a Don Juan-like compulsive change of lover). He also added, though only on the borderline of the present subject, that a reaction to guilt might take the form of an interest in religious and charitable organizations. In the child, said Dr. Rickman, guilt began early, and so did the considerable cultural restraint of its aggressive and other asocial impulses. Certainly by the time the child was 4 years of age it was a cultural unit, though not yet a stable one. "A child needs very little teaching about what is right and wrong; it is greatly helped if it is left to find its own way to a good relation even to not-so-good parents. A parent's failure to recognize the child's capacity for guilt is a failure to recognize that it is a human being."

The chair for the general discussion was taken by Dr. H. CRICHTON-MILLER, who spoke of the relevance of this subject to world peace. The behaviour of racial and national groups depended upon the values which were accepted by their leaders. There was a time when civilization stood for integrity in social and personal relationships, or at least that situation was being approached. Now the human race was facing starvation and possible destruction largely because leadership had fallen into the hands of men who put self-interest and expediency before integrity. How far could mental health in the individual citizen secure integrity of leadership? How far could truth and honesty be reinstated by the improved mental health of the citizen? Certainly to some extent, but equally certainly not in full measure; and there must be an external norm to which personal and communal values were to be related. Psycho-analysis had taught them that the old parental values, passing down by heredity, identification, and fixation, led to serious impairment of spiritual freedom, and, as they were all agreed, it was on spiritual freedom that democracy was based.

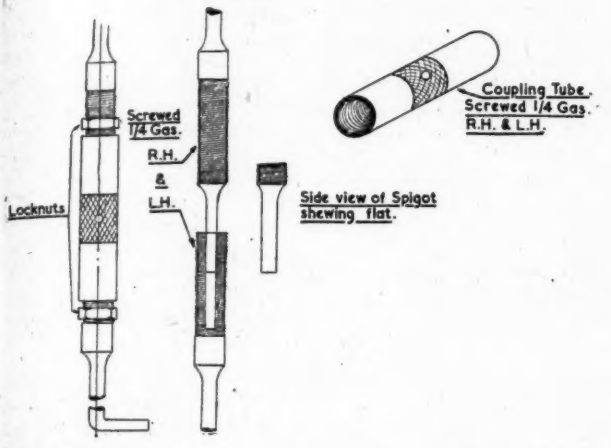
Dr. VAN DER HOOP (Amsterdam) opened the discussion, and was followed by Dr. CLIFFORD SCOTT (London), who said that his experience was that even in the most ill patients there had been moments of pre-ambivalent love just as there had been moments of pre-ambivalent hate, and that there was much capacity for love even in the most guilty. Other speakers were Dr. WALTER FURSTENHEIM (Great Britain), Mrs. MELANIE KLEIN (Great Britain), Dr. J. FLESCHER (Italy), and Dr. DEBROWSKI (Poland). Dr. W. C. HULSE (United States) pointed out what he considered to be a significant difference between the American and German soldier who broke down under the impact of war. The American soldier asked, in a large majority of cases, "What have I done in this conflict that I ought not to have done? I have killed a man, or I have given orders to others whereby men have been killed." There were an enormous number of neurotic disturbances of that kind. On the other hand, the German soldier in a similar situation did not feel guilt of that kind, but he would ask, "What have I not done that my superior asked me to do?" He was occupied continually with his relation to his superior officer, the father-figure, whereas the American soldier in his psychological breakdown revealed a quite different guilt pattern. Much more attention should be paid to the cultural factor which played an important part in the mobilization of feelings of guilt.

[To be concluded.]

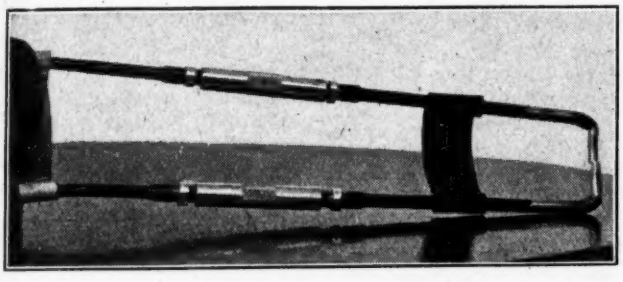
Preparations and Appliances

THE STRETCHING SCREW CALLIPER

Mr. HORACE DAVIES, visiting orthopaedic surgeon, Birkenhead Municipal Hospital and Whiston County Hospital, writes: It is with some trepidation that I describe the following modification of the time-honoured walking calliper. Adjustment of calliper length to individual comfort and requirements by the conventional sliding bar and lock-screw principle is not a precision



method although mechanically very sound. The principle that I have applied for the adjustment of calliper length is that of the stretching screw commonly used for cable-tightening. From the diagram it will be seen that the calliper side-steels are divided and coupled about 10 in. (25 cm.) above the extension ends. The coupling is by means of a left- and right-hand



threaded tube which screws over corresponding left- and right-hand threads in the side-steels. To prevent rotation the end of the top steel is shaped into a thin flat projection which slides into a corresponding slot cut in the end of the lower steel. By turning the coupling with its milled centre adjustments can be made easily and with precision. The required position is then set by the lock-nuts.

Continuing the series of reports summarizing the results of clinical surveys of the nutritional state of certain sections of the community, E. W. Adcock, H. E. Magee, E. H. M. Milligan, and H. S. Townsend describe in a recent issue of the *Monthly Bulletin of the Ministry of Health* (1948, **7**, 153) the results of investigations undertaken between February, 1947, and May, 1948. Representative samples of three groups were surveyed—adolescents, housewives with adolescent children, and school-children. In none of the groups was any frank deficiency disease observed, and in the adolescents and school-children the nutritional state was well maintained as compared with past years. The assessment of the state of nutrition of housewives, from the results of the examination of the 652 women who agreed to be medically examined, proved difficult: 87.4% were considered to be of good, 10.9% of fair, and 1.7% of poor nutritional status. In general it may be said that this group of mothers, with children aged from 12 years upwards, appears to be the most vulnerable section of the population yet examined.

Correspondence

The B.M.A. Under Fire

SIR,—When the B.M.A. Study Groups began, about six years ago, I was a member, and I well recall my astonishment that no discussion was allowed on terms of service. Since then, as general practitioner and consultant, I have argued with the Divisional Executive Committee of which I have been chairman, and which includes a member of Council, that clear conceptions of terms of service were essential to the B.M.A. and that without them no real hold on the body of the profession was possible. My arguments were futile; the official bodies of the Association stuck to general conceptions, the fundamental point at issue was made refusal to enter a whole-time salaried service, this was put to interminable meetings of the profession. It may be that the B.M.A. never imagined the granted this all necessary was won, and then the Minister agreed. He agreed, not to any proposal made direct to him by the B.M.A., but to a proposal made by the Royal College of Physicians, who put in their resolution no less than had been said by innumerable speakers at innumerable meetings of the profession. It may be that the B.M.A. never imagined the Minister would give an amending Act, and made that the cover for a demand for further amendments; it may be that they have not the essential ability to bring the enormous weight of opinion they undoubtedly accumulated to bear on the one man who mattered at the time that mattered on the issue that mattered—in other words, they lack a negotiator. At any rate, when the Minister gave way, the B.M.A. was left with no practical policy; it had never allowed itself to consider terms, and was left hopelessly in the air. A negotiator must know what he wants, or he cannot begin to serve his party. Hence the impotence of the B.M.A. and the sense of frustration of its members. Hence the deplorable present situation in which regulations are pouring out from overworked Civil Servants who know nothing of practical detail, instead of the situation we idealists had pictured in which the B.M.A. would have had plans ready in advance of the Ministry, instead of protests several weeks too late.

To any consultant who has watched all this the present situation is alarming. It should be pointed out that the B.M.A. Consultants and Specialists Body is nominally autonomous and its decisions are "not to be subject to approval of the Council or the Representative Body except in so far as they might affect other forms of practice or other aspects of the policy or activities of the Association." This is not autonomy at all. Every man who enters consulting work leaves general practice at some stage of his career, every pound that goes to consultants deprives the general practitioner or will do. Every rise in the standard of consultant or specialist practice will put a few general practitioner-consultants out of consulting practice. And every decision on these and similar fundamental matters the B.M.A. Consultant and Specialist Body makes will be liable to veto by the Council or Representative Body. Of course, the B.M.A. is not clear on the matter; at a recent regional meeting of the consultants a B.M.A. official told those present that the meaning of the Cambridge resolution was not clear to him, but added, "We understand it at B.M.A. House." How can this serve any useful purpose? What confidence can be felt in such a statement? It is the old situation over again: leave things to the B.M.A., who know, who negotiate, who have the machinery. The result seems likely to be the same unless the B.M.A. is very careful. The disillusion of the general practitioners will be equalled only by that of the consultants. And what clear conceptions of a consulting service has the B.M.A.? Can it claim to know more than the professors of universities and deans of medical schools? How many new representatives better than these experienced men is the B.M.A. prepared to produce? And where is it going to find them if not in the Colleges?

This letter is written by a member of the B.M.A. who has served it locally to the best of his ability, and who has seen many of his worst forebodings proved miserably true. It is an unfortunate thing that honest criticism is called disloyalty, when

it is not regarded as an abstruse form of mental defect or emotional displacement. But one more effort seems called for, and has been made. Can the B.M.A. not grant the consultants and specialists true autonomy—dominion status? And can they not recognize the vital part the Colleges must play, that academic and financial matters are no longer separable, and that for the good of all the leading consultants should be recognized as leaders?—I am, etc.,

Hove, Sussex. W. A. BOURNE.

Paratyphoid Osteomyelitis

SIR,—The two examples of paratyphoid B osteomyelitis reported by Drs. Rachmiel Rozansky, E. N. Ehrenfeld, and Y. Matoth (Aug. 7, p. 297) prompted me to read my notes of a similar probable, though bacteriologically unproved, case of this rare condition. Briefly, this was a girl of 16 admitted to Chase Farm Hospital in 1942 with pyrexia and pain in the lower back of one week's duration. Osteomyelitis of the spine was considered, but investigation showed that she was suffering from paratyphoid B. No radiological signs were present while the pyrexia lasted. A tentative diagnosis of early ankylosing spondylitis was made, and she was put in a plaster bed for three months. *X*-ray examination then showed complete destruction of the body of the fifth lumbar vertebra. Further immobilization resulted in a stable and symptomless spine. She was followed up until 1945, and I saw her in the street last year apparently quite well.

The interval of 32 years between the enteric fever and bone abscess in Dr. Rozansky's second case warns me that although bone destruction has occurred my patient may not be out of the wood yet.—I am, etc.,

London, N.14. C. ALLAN BIRCH.

Who Shall Minister to the Neuroses?

SIR,—Is it fully appreciated that Dr. C. A. H. Watts has rendered a great public service by his unassuming article on this subject (July 24, p. 214)? For he demonstrates therein that sufferers from the anxiety state can be very helpfully treated psychologically on simple common-sense lines by a general practitioner.

It is admitted that about a third of our chronic patients are solely in need of psychological treatment of a kind which the average doctor has neither the time nor the education to provide, and only a minority have the necessary flair. His ordinary fees do not reward the extra time involved, and he naturally dislikes to surrender his influence over his patient by invoking a psychological specialist who is needed for complicated cases. But most cases can be treated on simple lines as Dr. Watts has shown.

I see no solution of this pressing problem until a considerable proportion of general practitioners have been trained to treat the body-mind—recognized by a later degree (perhaps an M.D.) and rewarded by higher fees. The M.B. only qualifies to treat the body. Ordinary examinations test knowledge rather than the ability to use it. Special methods would be needed to detect the flair needed in this treatment. To treat the body-mind can scarcely be taught in lectures but can be learnt (in a few years) from patients, with the help of books such as *The Common Neuroses* by T. A. Ross (1937).

I speak from experience. Many years ago I realized as a general physician that it was necessary to treat the body-mind on such simple common-sense lines as Dr. Watts advocates. Complex cases were beyond my scope, but they were a minority. This deplorable hiatus in treatment must be filled. To produce enough general practitioners of the right type will need long-term planning on a national scale. Dr. Watts has shown that it is possible.—I am, etc.,

Beverley, Yorks. F. C. EVE.

Use and Abuse of Tonsillectomy

SIR,—Mr. T. B. Layton's reference (Aug. 7, p. 310) to the prohibition of tonsillectomy during the poliomyelitis epidemic last year brings up a very important matter—namely, the necessity or otherwise of such a step. As the prohibition was not absolute, a good many surgeons must be in a position to quote figures of cases done during the epidemic. I personally did nearly 500 cases without anyone developing the disease, and conversation with colleagues leads me to believe that my experience was not exceptional.

Statistics from other countries point to an increased liability to poliomyelitis after recent operations on the upper respiratory tract. But is this increased susceptibility sufficiently great to warrant the prohibition of such operations? No doubt statistics, if such were available, would show an increased liability to all diseases involving the upper respiratory tract after recent operations on this region, so logically such operations should stop during practically all epidemics. This may well result in tonsil and adenoid operations ceasing for a large part of the year. If the contention that the number of such operations is excessive is correct, this may be a blessing in disguise. But is it correct?

In spite of discouragement from the Ministry of Education doctors and nurses continue to advocate, and parents to demand, the operation in a large number of cases. Whether this number is excessive, or in other words includes an appreciable number of tonsils which are normal or likely to return to normal, is a matter which cannot be decided without scientific research. I therefore heartily endorse Mr. Layton's statement that the profession as a whole should reconsider its approach to tonsillectomy, and I think we would be justified in looking to the Medical Research Council for a lead in this matter of great practical importance.—I am, etc.,

Birmingham, 15. ROBERT EVANS.

SIR,—I should like to support the views expressed by Mr. T. B. Layton (Aug. 7, p. 310). Neglect of chronic nasal catarrhs causes, as in measles too, infection of the middle ear. Damage having been done, removal of tonsils for their further treatment can do no good. One of the complications following tonsillectomy is middle-ear disease. Diastolization can cure most nasal catarrhs, or, if taken early, nasal oil may be successful. Sinusitis leading to enlarged adenoids can be relieved by Proetz's displacement method. Looking only at tonsils cannot decide the need for the removal of adenoids. I think the decision should be left to the parents, and doctors should not dictate to them as to necessity. Dr. J. Alison Glover's survey of 90,000 tonsillectomies showed the rate of operation varied from 1% to 45% in different counties, and at one school the number increased with the income of the parents. Where the operation was reduced in number the so-called "dangers" of so-called "septic tonsils" did not exist. The consultant aurist at the Manchester health department has shown the value of treating nasal catarrhs and the reduction of tonsillectomies.—I am, etc.,

Blyth. A. G. NEWELL.

SIR,—I have read with great interest the letter (Aug. 7, p. 310) by my old friend Mr. T. B. Layton on the use and abuse of tonsillectomy, and I congratulate him on an excellent and reasoned exposition of this subject; which is, especially to general practitioners, a most interesting and often worrying problem.

As he so wisely says, "We cannot conduct the art of medicine by administrative fiats." I am convinced that many tonsils and adenoids are removed unnecessarily, and I have seen many bitter disappointments in parents at the unrealized though expected improvement in their children after this operation. The converse equally holds good, and I have seen many a child steadily regress in health owing to the parental refusal of an obviously necessary operation. Following a sore throat many tonsils are removed owing to mere enlargement, through the failure to realize that this hypertrophy is only physiological and temporary. In my opinion the two pointers for operation are (1) glandular enlargement plus the fact that pus is exuded from the tonsil on pressure, and (2) obstruction to the air intake, most often but by no means always due to adenoid vegetations, the patient showing mouth breathing by night with consequent chest deformities.

Much has been written on symptomatology, but my submission is that, epidemic or no, each case must be judged on its individual merits.—I am, etc.,

London, S.W.1. DESMOND MACMANUS.

Thyroidectomy Fatality Rate

SIR,—In his comparison of safety between thiouracil and surgery for thyrotoxicosis, Professor H. P. Himsworth states (July 10, p. 61) that "it is probable that the mortality rate (after surgery) in the best general hospitals, both in this country and abroad, is not far below 2%." I cannot help thinking that this is a most pessimistic estimate for modern thyroid surgery. For a period of 29 years I have had the honour to anaesthetize for three surgeons who specialized in thyroid work, and for the whole period the total operative mortality rate for thyroidectomy was 0.75%. This includes all patients who died in the hospital or nursing-home.

In recent years, however, owing to the advances in preoperative treatment and in surgical and anaesthetic techniques, the mortality rate has fallen to a very low figure, there having been only two deaths in the last consecutive 1,000 cases (0.2%). There is no reason to suppose that this figure differs appreciably from that obtaining generally, and I would suggest that the insertion of a decimal point in Professor Himsworth's estimate would give a more accurate idea of the present-day mortality rate from thyroid surgery. I should, perhaps, add that the majority of the thyroidectomies referred to were performed at St. Bartholomew's Hospital either in London or at Hill End.—I am, etc.,

St. Albans, Herts. C. LANGTON HEWER.

Prevention of Dust Diseases of the Lung

SIR,—From this correspondence (July 17, p. 172, and Aug. 7, p. 311) it would appear that so far we do not possess a face mask or respirator which in use will efficiently arrest dangerous dust particles and assure the workman of protection against pneumoconiosis. At the same time it seems agreed that face masks do not represent effective action directed to the control of the disease. From practical experience one thing is certain —namely, that no mask has yet been devised which heavy manual workers, particularly workers at the coal-face, can comfortably wear throughout a day's work.

As a temporary expedient practical scientists are seeking to design a mask which is efficient in arresting the dust and at the same time is acceptable to the workman. To those scientists I would suggest they have a further responsibility, which is to ensure that the wearing of the mask, its day-to-day storage, and disposal of pads do not bring new dangers. Workmen and employers are exceedingly careless in what to medical men are elementary matters of hygiene. It is a common observation at workplaces to see masks still fitted with soiled pads thrown into a common receptacle, from which masks are taken promiscuously when required, while soiled infected pads are discarded anywhere. The recent article by Dumbell, Lovelock, and Lowbury (*Lancet*, July 31, p. 183) on "Handkerchiefs in the Transfer of Respiratory Infection" may not be entirely irrelevant to this aspect of the problem, especially as it has frequently been argued in the literature that respiratory infection by pathogenic organisms may modify adversely the action of noxious dust in the lungs.

This is no place for a discussion of measures for the effective control of pneumoconiosis in coal-mines, but I would venture to ask your correspondents, Dr. A. Harper and Dr. J. M. Morgan (Aug. 7, p. 311), or any others, what is the *earliest harmful evidence* in coal-miners at which the workman should be advised to give up his skilled occupation and seek alternative work? Or, in the language of the compensation schemes, "What constitutes the disease to a *dangerous* degree?" This is the question which more than any other urgently demands a clear, unequivocal answer now.—I am, etc.,

Glasgow. A. MEIKLEJOHN.

Fibrositis

SIR,—The article on fibrositis by Dr. James Cyriax (July 31, p. 251) certainly offers a simple solution to an aged and complex problem. The conclusion that he "forced on the unprejudiced observer" that "the symptoms of rheumatic fibrositis coming on for no apparent reason are the result of articular lesions," and "the underlying principle of treatment" that he wishes to teach the simple observer, that of the reduction of the intra-articular lesions by local manipulation, are simple if not factual. It would be interesting to know if Dr. M. Woodhouse is as happy about the finality of the conclusions drawn from his necessarily limited electromyelographic studies as is Dr. Cyriax.

It is an accepted fact that many people who in the past have been diagnosed as suffering from fibrositis and neuritis are really suffering from symptoms due to intra-articular lesions such as prolapsed and fragmented intervertebral disk in the cervical or lumbar regions, and that the principle of local treatment is either movement or rest. Dr. Cyriax has been among those who have impressed this fact on the medical profession. This, however, is not the problem of "fibrositis," and in consequence the solution offered in this article is no solution.

"Fibrositis" is a clinical term commonly used to describe the pain which seems to be located in the soft tissues of the limbs and for which there is no obvious cause. In practice the diagnosis is based on the exclusion of known systemic diseases or local disorders. Pain and stiffness are the cardinal symptoms, occurring particularly on movement and localized on clinical examination to the subcutaneous tissues, superficial and deep fasciae, aponeurosis, ligaments and capsules of joints, tendons and tendon sheaths, bursae, periosteum, and the sheaths of the nerve trunks.

It is true that we have no adequate evidence that fibrous tissue is affected primarily, but we do know, many of us from personal experience, that sitting in a draught may be followed by a stiff neck or lumbago; also we know that pain in the shoulder may disappear after the removal of a septic tooth. We do know that the industrial worker, the harried young housewife, and the plump, middle-aged ladies among our acquaintances frequently suffer from "fibrositis." Many of us know that the "reduction of an intra-articular lesion" has not only failed to relieve their symptoms but has made them worse.

"Fibrositis" is a problem in general medicine. At the present time "unprejudiced" and far from simple observers are studying this problem, and I would bring to the notice of Dr. Cyriax and his would-be disciples the many papers and books on this subject recently published in the English language. I would mention only those by Weddell, the anatomist; Draper, the constitutionalist; May Wilson, the clinician and geneticist; Halliday, the industrial physician and psychiatrist; and Ling, the sociologist. Hench, Stone, and other physicians whose primary interest is in rheumatism have also made valuable contributions. Finally I refer Dr. Cyriax to the papers by Drs. Bauwens, Harman, and Dykes of his own hospital.

The problem of "fibrositis" is complex. To solve it the biochemist, the geneticist, the pathologist, and the clinician will need to work together as a team, probably for quite a long time to come.—I am, etc.,

London, W.1. FRANCIS BACH.

Treatment of Chronic Varicose Ulcers

SIR,—I read the paper by Messrs. John Borrie and E. Vernon Barling on chronic varicose ulcers treated by lumbar sympathectomy (July 24, p. 203) with great interest. When I was Mr. (now Professor) Ascroft's house-surgeon we had a patient suffering from thromboangiitis obliterans with large ulcers in the region of his malleoli which caused him much pain. To relieve this pain Mr. Ascroft divided the superficial nerves supplying the area in question, and to our surprise and gratification not only was the patient's pain relieved but the ulcers quickly became smaller and healed within a very short time. Mr. Ascroft attributed this effect to the improved local circulation, as the sensory nerves also carry some of the sympathetic fibres to the area.

Since then I often divided or crushed the great saphenous nerve just below the knee when I did a Trendelenburg operation in patients with varicose ulcers over the medial malleolus, and always had the impression that this simple manœuvre speeded the healing of the ulcer as well as relieved the patient's pain immediately.

The nerve is easily found a couple of inches below the knee, where it lies close to the saphenous vein, which I ligate in this place as well as in the thigh in these cases. Often the nerve is not in a single trunk but divided into several strands. In the case of ulcers in the region of the lateral malleolus the sural and the cutaneous branch of the superficial peroneal nerve require division. This little operation adds only a few

minutes to a routine Trendelenburg operation and is, in my opinion, always worth while doing before embarking on a major operation like lumbar sympathectomy.—I am, etc.,

Coventry. P. E. ROLAND.

Prevention of Venereal Disease

SIR,—I feel obliged to add my testimony on the subject of prevention of venereal disease to that of your correspondents in the *Journal* of July 31 (p. 268). I am sure that to rely upon methods of prophylaxis to be applied after exposure to infection is to court failure. The attention to detail necessary to make them efficient is rarely available, and the false sense of security given simply encourages risks to be taken. Dr. Earle Moore, with his vast experience, has no doubt that prophylactic treatment if properly carried out can be of value, but he says the average man will not take the necessary trouble; and when one remembers how often the patient was more or less under the influence of alcohol when the risk was taken this is not surprising. If anything is to be done on such lines of prevention, surely the use of a rubber sheath is much simpler and more likely to be carried out; this indeed is what I am in the habit of telling those patients who seem unlikely, in spite of all one can say, to abstain from future risks.

Finally I must add that I am astonished Lord Horder (July 17, p. 171) should take such a pessimistic view of the present situation. Looking back over the 25 years that I have been in charge of a fairly busy clinic I feel that, owing chiefly to the action of the Ministry of Health in making the public "V.D. conscious" and in providing free treatment, we can reasonably anticipate a continuation of the improvement which was so marked before the war and which is now showing itself again. That war conditions should cause a relapse was inevitable and to be expected. Has not this happened in every war since syphilis was first brought to Europe by soldiers in the sixteenth century?—I am, etc.,

East Croydon, Surrey. P. W. HAMOND.

SIR,—The sexual requirements of the individual vary immensely, and each is entitled to judge for himself or herself whether extramarital relations are right or wrong. There can never be a standard sexual morality. The problem is therefore a moral one for the individual only. To regard V.D. as a moral problem, as does Dr. G. L. Russell (July 31, p. 268) is to be prejudiced. It would appear from what Miss K. B. Hardwick writes (p. 269) that the Association for Moral and Social Hygiene, having found chastity as an ideal unsuccessful, is now preaching chastity as an anti-V.D. device.

The first essential in any campaign against V.D. is to stop looking upon extramarital relations as a disease in itself. The lack of a marriage certificate does not transform sexual experience from something beautiful into something ugly and shameful, but the present official outlook of Church and State, by driving sex into back streets and sordid lodgings, does precisely that, and at the same time favours the spread of V.D. The inevitability of extramarital sex relations must be accepted, and every effort made to keep such relations, if not on a spiritual, at least on a healthy physical level.

All anti-V.D. appliances should be banned until some really efficient method is discovered, and V.D. propaganda stopped. Would not anti-T.B. propaganda be of more value in its own field? Surely the early symptoms of pulmonary tuberculosis are more insidious than those of any of the venereal diseases, and early treatment more important.

Until some super prophylactic drug which can be taken by mouth comes along, legalized and controlled prostitution, as was carried on until recently in France, is the only sure method of V.D. control. The termination of this system in France has led to an increase in V.D. The relaxation of official harshness manifested in the adoption of such a system would also do much to establish a healthier outlook on sexual matters, and a more sensible attitude to V.D. A quotation from *Sex, Life, and Faith* by Rom Landau is appropriate, "The number of divorces for sexual misdemeanour is much smaller in France than in Great Britain . . . they have suffered far less from neuroses or sexual aberrations. Evidently their more frank and natural approach to the problem of sex is to some extent responsible."—I am, etc.,

Edinburgh. W. B. LAING.

Confidential V.D. Treatment

SIR,—By her complaint against the Minister of Health for removing the statutory requirement of secrecy about V.D. Miss Katharine B. Hardwick (Aug. 7, p. 313) fails to see the wood for the statutory trees and betrays signs of a legalistic mind out of touch with realities.

When in 1916 the V.D. service was started the Government accepted the recommendations of the Royal Commission on Venereal Diseases that attendance should be voluntary and free, but added a statutory requirement of secrecy which was not one of the Commission's recommendations. No doubt this addition was made for propaganda purposes to beguile an unprepared and possibly distrustful public into voluntary attendance at V.D. clinics. No doubt also the provision was inserted in deference to the fears of those who thought that the public would not regard public clinics as sufficiently private and that therefore V.D. would still be treated inefficiently in an underground manner by unqualified persons.

Whatever the reasons for inclusion of the provision of statutory secrecy there is no doubt that after more than thirty years of experience of the V.D. service the public is firmly convinced that the staffs of V.D. clinics do in fact maintain the greatest secrecy, and few members of the public who use the V.D. clinics are as well informed as Miss Hardwick about the legal background or as impressed by its power.

Having worked in V.D. clinics for many years I have no hesitation in emphatically stating that the high standard of secrecy and confidence obtaining in these clinics is due neither to statute (as she asserts) nor to Ministerial hopes (as she agrees), but to the sense of responsibility and loyalty of the medical and lay staffs, who have learned during more than thirty years' experience the great importance of securing the trust and confidence of their patients. Secrecy is now so strongly entrenched by custom in the minds of public and clinic staffs alike that a statute is unnecessary to buttress it. Rather than complain that a Minister of a Socialist Government revokes regulations we should congratulate him and those who advised him for removing unnecessary legal lumber.

Finally, by encouraging ventilation of the Minister's revocation of statutory secrecy Miss Hardwick is tending to obtain the very opposite of what she wants. If she says from the mountain tops that there is now no secrecy about V.D. (because it is not statutory) not only is she publishing error, not only is she casting an unwarranted slur on the devoted staffs of V.D. clinics, but she is herself likely to cause unfounded doubts to arise in the minds of the very public she wishes to protect.—I am, etc.,

London, N.W.5. F. R. CURTIS.

Thiouracil in Treatment of Thyrotoxicosis

SIR,—We should be grateful to Professor H. P. Himsworth (July 10, p. 61) for his lucid exposition of the status of thiouracil in the treatment of thyrotoxicosis. For more than a year I have taught that white counts are not a routine necessity in thiouracil therapy, but that they should be done in the three following circumstances: (1) In the first few weeks of treatment they are valuable in order to detect the occasional case in which granulopenia reaches a dangerous level. It is helpful to know the usual white cell level of each patient, for this varies greatly in different individuals (my own white count is usually between 4,000 and 5,500 with about 50% to 60% of polymorphs), and it is correspondingly difficult to generalize regarding the level of granulopenia. (2) A white count should be done whenever the minor toxic effects of thiouracil appear. By themselves these manifestations do not call for cessation of the drug; it is the level of the white count that matters. (3) Agranulocytosis: I fully agree with Professor Himsworth that routine white cell counts are no safeguard in preventing or detecting this dangerous complication.

I believe that the quickest way of dealing with a suspected case of agranulocytosis is to make a thick film of peripheral blood, as is done in the diagnosis of malaria, and stain it with Field's stain[1]—a few seconds' gentle agitation in solution A, then in distilled water, then in solution B, and finally in water again. Anyone can make a good thick film if they trouble to practise, and most people should be able to distinguish a polymorph in a thick smear. Given a microscope and the necessary stain (which

can be carried in screw-capped bottles wide enough to admit a microscope slide) the method should be within the scope of any competent practitioner).—I am, etc.,

F. J. ZACHARIAS.

Liverpool.

REFERENCE

1 Field, J. W., *Bull. Inst. med. Res., Federated Malay States*, 1941, No. 2.

Dietetic Treatment of Hepatic Cirrhosis

SIR,—In the leading article under the above heading (June 5, p. 1089) you suggest among other considerations the use and clinical test of antithyroid drugs in the treatment of human hepatic cirrhosis. It is a pleasure for us to let you know briefly about our work in this subject since 1946. We used thiouracil in 11 cases of human cirrhosis based on the works of György and Goldblatt (*Science*, 1945, **102**, 450). These authors demonstrated that the addition of thiouracil in the cirrhosis diet for rats plays a powerfully preventive role. In our group of patients the drug was used in amounts of 0.6 g. per day with a total dosage which fluctuated between 3.6 g. and 20.4 g.

In order to estimate the effects of this treatment three groups of cirrhotic patients of the same condition were studied—one group (the control) with hydrocarbonate diet, glucose, serum, and paracenteses; a second with a hyperprotein diet and vitamin B complex; and a third with hyperprotein diet, B complex, and thiouracil. The results of these different systems of therapy are summarized in the following table.

Group	No. of Cases	Better	Results Same	Worse
I	9	1 (11%)	4 (44·5%)	4 (44·5%)
II	17	7 (41%)	4 (24%)	6 (35%)
III	11	7 (63·6%)	1 (9%)	3 (27%)

Those requiring further information may like to refer to our paper in *Jornadas Clínicas de Verana, Chile* (1947, **2**, 291).—We are, etc.,

C. SILVA LAFRENTZ.
D. VERDUGO BINIMELIS.

Viña del Mar, Chile.

Liquid Paraffin in Baking

SIR,—In the leading article entitled "Liquid Paraffin Risks" (June 12, p. 1141) mention is made of the potential dangers inherent in the use of medicinal paraffin and other less refined mineral oils in baking. The work of Frazer and his colleagues indicating the possibility of liver and other damage due to mineral-oil absorption when ingested as finely dispersed emulsions of particle size less than 0.5 μ is reported, and comment is made on the lack of information regarding mineral oil distribution when dispersed in flour products, such as cakes and pastry, baked with liquid paraffin as a replacement for vegetable fat.

We have recently examined some emulsions containing mineral oil before and after heating, the unheated emulsions containing particles ranging in size from less than 0.5 μ to 10 μ or greater. On heating, the individual oil particles coalesce, forming larger aggregates, until ultimately the emulsion is completely broken into two layers. It might be anticipated therefore that, after incorporation of liquid paraffin in a cake batter, on baking the emulsified material would break down and no particles would remain in the final product less than 0.5 μ in size. This supposition is not, however, in accord with the observations herein reported. Examination, using a Reichert optical system, of sponge-batter smears containing liquid paraffin, baked in a micro-oven (to be described elsewhere) and under continuous observation on a glass screen, indicated that although some aggregation of the initially finely dispersed liquid paraffin did occur there still remained in the final product some particles approximately 0.5 μ in size. The smears under examination were taken from a bulk batter composed of cake flour 14 g., sugar 10 g., dried egg 3 g., baking powder 1 g., water 10 ml., and liquid paraffin 15 ml. A red, oil-soluble, water-insoluble dye was used to colour the liquid paraffin in order to differentiate it from the other constituents of the batter. Magnification was 600 diameters and the observation screen had a scale etched thereon so that direct observation of particle size could be made.

In the experiment quoted some 30% of the total number of liquid paraffin particles present in the uncooked batter were about 0.5 μ in size, and although this percentage was considerably reduced after baking some very small particles were still clearly discernible. It should be noted that the actual weight percentage of the original mineral oil used that would exist in this fine state of subdivision would be extremely small, and it might be claimed that even on continuous ingestion over long periods absorption of sufficient oil likely to cause liver damage might not occur. Nevertheless the very existence of a number of small particles which come within Frazer's classification as potentially liable to cause liver damage suggests the desirability of ascertaining whether there exists a threshold limit to their number before damage becomes real.

The work of Alvarez[1] indicates, however, that the fine emulsification necessary for liver damage can be effected by the bile acids, oleic acid and glyceryl monostearate, which may be found in the bowel. In this connexion it should not be overlooked that glyceryl monostearate is a common emulsification agent and is, in fact, now used in the preparation of certain foods. It would appear therefore that, even though the number of particles of mineral oil of size less than 0.5 μ appears to be considerably reduced on baking, this may be immaterial inasmuch as subsequent ingestion may well restore the level of such particles to the original value. Further physiological investigation on these points is clearly desirable, as the problem would appear to be of even wider significance than the editorial previously referred to implies.—We are, etc.,

J. B. M. COPPOCK.
R. C. A. BRADSHAW.

The British Baking Industries Research Association, Chorleywood, Herts.

REFERENCE

1 Alvarez, W. C., *Gastroenterology*, 1947, **9**, 315.

Comprehensive Child Health Service

SIR,—Dr. H. R. Youngman's letter (Aug. 7, p. 313) stirs me to comment on the recent correspondence on this subject. A high percentage, I should say the majority, of complaints for which the child is brought to the doctor these days are described as behaviour problems—e.g., feeding problems, sleeplessness, bed-wetting, etc. These are usually the result of home conditions and parental mismanagement. How can a "clinic" doctor appreciate these conditions unless he has an intimate knowledge of the complete home picture and of *all* its members? To abolish the general practitioner would be to kill the root and main stem of medicine—its specialist branches would then soon wither and die.

What we want are more general practitioners with better undergraduate training in paediatrics, who have time to know and understand the home life of their patients so that with the aid of improved housing conditions and social services behaviour problems can be more easily corrected or even prevented. Then we shall find the paediatric specialist will be superfluous.—I am, etc.,

C. ELAINE FIELD.

London, N.W.7.

Geriatrics

SIR,—The first supplement to the report of the B.M.A. Committee on the Care and Treatment of the Elderly and Infirm (Aug. 7, p. 71) contains several interesting recommendations for the solution of the chronic-sick problem. However, one important point seems to have been overlooked—this is the need to train more doctors in the special techniques which have been evolved by such hospitals as the West Middlesex, Orsett Lodge, and St. John's, Battersea. It is probably true to say that a hemiplegic patient treated by Dr. Marjory Warren's methods has a better chance of complete recovery than one not so rehabilitated. A patient with incontinence of urine is just another bed-wetter in hospitals where Dr. Thomas Wilson's cystometric technique is unknown. A chronic arthritic treated by Mr. Grant Waugh's procaine–lactic acid injections has prospects of improvement undreamt of by those who have not used this method.

In the same way the rehabilitation of elderly patients can reduce the number of bedfast patients considerably, as mentioned in the report.

Why does the Ministry of Health take no steps to make this special knowledge available throughout the country? The discoverer of a new form of treatment for cancer would probably be given every facility for making his techniques known

all over the world. Yet in this country the chronic-sick problem is even more pressing. General practitioners sometimes have to strain the truth in order to get their elderly patients admitted to hospital. Nursing staff avoid "chronic" hospitals in which active treatment followed by recovery is unusual. A training school of geriatrics would not be likely to suffer in this way from shortage of staff. We have had many hospitals for children, and now there is a postgraduate institute of paediatrics where specialists in this branch of medicine are trained. What about a hospital for the elderly and a postgraduate centre for geriatrics?—I am, etc.,

Purley, Surrey. TREVOR H. HOWELL.

Medicine in General Practice

SIR,—It would be interesting to know if Dr. D. V. Hubble, who reviews (Aug. 7, p. 301) Dr. R. P. McCombs's *Internal Medicine in General Practice*, is himself engaged in general practice. He finds fault with the book because only half a page is devoted to cerebral tumour. I can recall only one case of intracranial tumour encountered in twenty years as a G.P. This baffled the diagnostic acumen of at least one consulting surgeon and one consulting physician. Another consulting physician suspected the condition. He referred the patient to a neurosurgeon, who failed to localize the tumour after full investigation at a special clinic. I sent the man to a hospital of international fame, where he lay for months before a sufficiently accurate diagnosis to permit operation could be made.

It seems strange to me, as a general practitioner, to expect a textbook of this kind to be devoted to any more detailed consideration of diseases of the nervous system than can be encompassed in one-eighteenth of its volume. The vast majority of these diseases are incurable, and all the G.P. can usually do is to ask a consultant to announce the verdict to the patient. Certainly diseases of the nervous system do not form one-eighteenth or even one one-hundredth of our daily work.

There may be areas in this country where the cost of *x*-ray investigation has not hitherto precluded its use as a "nearly routine measure" in gastro-intestinal disorders, but it would be by no means true of the country as a whole. The G.P. must in the first instance refer his poorer patient to the appropriate out-patient department, where the physician or surgeon in charge decides as to the necessity or otherwise of *x*-ray examination. One greatly doubts if the dawn of the Bevan era means that the G.P. will be able to use full *x*-ray study as a nearly routine measure for his patients.—I am, etc.,

Launceston, Cornwall. DONALD M. O'CONNOR.

POINTS FROM LETTERS

Service Doctors in Far East

Lieut.-Col. LENNOX R. S. MACFARLANE, R.A.M.C. (Tripoli), writes: I was amazed at the unfairness and onesidedness of Lieutenant D. R. Morgan's letter (July 3, p. 54). In fairness to your readers let me put the other side of the question. The "regular" who has no "difficulty in obtaining a passage" has to wait in any foreign station at least six months before he can get his family out and live "in comfort if not luxury." Moreover, this happens at frequent intervals and will continue to do so while there is a shortage of quarters, and this is in addition to the many years' separation most R.A.M.C. regulars had to go through during the war. Also when the regular goes home for a short spell he goes to a station where there is another six months' delay in finding a house. If the "conscript," as Dr. Morgan chooses to call him, were to have equal rights in quarters and passages he *and* the regular would have to wait at least a year to get their families out, which would be most unfair to the regular who has taken the job on voluntarily and of little use to the conscript who is only abroad for 18 months. . . .

Fibrositis

Dr. CYRIL HELM (Plymouth) writes: I was interested to read the article by Dr. James Cyriax on fibrositis (July 31, p. 251). When I came to Plymouth about a year ago, I was astonished to find that a large number of my patients were suffering from fibrositis or muscle spasm, call it what you will. Some of the cases were due to articular displacements, but the majority definitely were not. The climate here is humid and muggy, and this must have some bearing on the matter. A fair proportion of these cases are women who are tired and anxious. . . . Physiotherapy and drugs are of little avail, but a good holiday or a week in bed, with plenty of sleep, cures the majority of them. . . .

Obituary

JOSEPH CUNNING, M.B., F.R.C.S.

Mr. Joseph Cunning died at his home in Reigate on July 29 at the age of 76. He was senior surgeon at the Royal Free Hospital from 1919 to 1931.

Joseph Cunning was a son of the late James Erskine Cunning, and he was born in Victoria, Australia. He was educated at Ballarat, the University of Melbourne, and St. Bartholomew's Hospital, London. His association with the Royal Free Hospital began in 1901 when he became the senior resident medical officer; in due course he was elected to the honorary staff, and in 1919 became the senior surgeon of the hospital, which position he held until his retirement in 1931. He was also surgeon to the Royal Cancer Hospital and the Victoria Hospital for Children, and a president of the Australian and New Zealand Association of Medical Men in England.

Mr. Cunning was a man of great personal charm, kindly and debonair, and he filled the post of senior surgeon to the Royal Free to the enhanced reputation of the hospital. He was wise in counsel, tolerant in judgment and criticism, and possessed of a kindly wit and great sense of humour which endeared him to his colleagues. His surgery was an art and a delight to watch—he was always cool and imperturbable, bold in design and skilful in execution. He was a great teacher, always ready and willing to help his juniors by advice, example, and assistance. There are many surgeons to-day who owe their skill and experience to Mr. Cunning's unfailing kindness and help, and they will remember with gratitude their debt to him.

Although Mr. Cunning was a general surgeon, his chief interest was in the surgery of the upper abdomen. In the days when gastric surgery was in its infancy he was, as he himself expressed it, an ardent gastrectomist. He was the author of many papers on surgical subjects. The first three editions of *Aids to Surgery* (from 1904 to 1913) were from his pen.

After his retirement from the Royal Free Hospital Mr. Cunning gave up surgical practice completely and retired to Broome Park, Betchworth, Surrey, where he transferred his interest and skill to the cultivation of irises and other gardening activities. Mr. and Mrs. Cunning in 1946 gave this lovely house and grounds to the Electrical Industries Benevolent Association as a home for old people, to serve as a memorial to their son, James Erskine Cunning, who was killed in action in 1941. Mr. Cunning is survived by his wife—herself a medical woman and a Royal Free Hospital graduate—and by two sons and one daughter, who has followed in her parents' footsteps. To all of them sincere sympathy will be extended by all who knew and loved Mr. Cunning.—G. B.

Miss K. G. Lloyd-Williams writes: Past students of the London (Royal Free Hospital) School of Medicine for Women have heard with sorrow of the death of Joseph Cunning. We knew him as a first-class surgeon and teacher, and his early retirement from surgery in 1931 was greatly regretted. His punctual arrival in the quadrangle, immaculately dressed, with buttonhole and cigar just at its last inch, was the prelude to a round in which in an apparently informal way he imparted a wealth of information. His humorous anecdotes enlivened the afternoon and served to impress salient facts on his audience. In his own inimitable way he was in the first rank of teachers. A versatile and amusing companion, he was the ideal mentor for young residents, who learnt much from his humane outlook on life.

As an operator he was outstanding, more especially in the field of abdominal surgery. The first to acknowledge and appreciate skill in others, one of his most engaging qualities was his modesty about his own work. He gave no appearance of haste or showmanship, but his results testified to his high skill. He was deeply attached to the Royal Free Hospital, and its welfare was always very near to his heart. He gave the hospital service of the highest order, and we all remember him with affection and pride.

W. C. ALLARDICE, M.D., F.R.C.S.Ed

We announce with regret the death at the age of 77 on Aug. 2 of Dr. W. C. Allardice, honorary consulting surgeon to the North Staffordshire Royal Infirmary.

William Clachan Allardice was educated at Bon Accord, Aberdeen, and Glasgow University. He qualified M.B., C.M. in 1892, and took his M.D. of Glasgow four years later; in 1906 he took the F.R.C.S. of Edinburgh. After qualification Allardice was house-surgeon at Macclesfield General Infirmary, and subsequently house-surgeon and then house-physician at the North Staffordshire (now Royal) Infirmary. At one time he was resident clinical assistant to the Montrose Royal Asylum. At the turn of the century Dr. Allardice went into partnership at Newcastle-under-Lyme with the late Dr. G. S. Hatton, and remained in the same practice until he retired in 1945. In 1902 he became honorary assistant surgeon to the North Staffordshire Royal Infirmary, and subsequently honorary surgeon and orthopaedic surgeon to the same institution. When he retired from the active list under the infirmary's age regulation in 1930 he became honorary consultant surgeon. For many years he was also consulting surgeon to the Congleton War Memorial Hospital, and during the war of 1914–18 was surgeon to the London Road Military Hospital at Stoke-on-Trent. Dr. Allardice contributed a number of articles to the medical press, his earliest being one to the *Glasgow Medical Journal* in 1896 entitled "Notes on 1,500 Cases of Anaesthesia." He joined the B.M.A. in 1895, was a representative in 1922, and president of the Staffordshire Branch 1926–7; he was also chairman of the North Staffordshire Division 1933–4. Dr. Allardice had a successful career as a general practitioner surgeon, and it was due to him that the Orthopaedic Department and the Ear, Nose, and Throat Department were established at the North Staffordshire Royal Infirmary. By his first wife, who died in 1903, he had one son, and by his second wife, whom he married in 1911, he had two sons and two daughters. He won the affection and esteem of those among whom he lived and worked, and inspired confidence in his patients and respect among his colleagues as a man who gave of his best to his chosen profession. He was a justice of the peace for Newcastle-under-Lyme, and was at one time Mayor of Hanley. A large gathering paid their tribute to his memory at the funeral held at St. George's Church, Newcastle.

R. A. K. writes: Bill Allardice, as he was affectionately known by a host of friends, qualified from Glasgow University at the age of 21. He was a most able and conscientious surgeon and had developed a clinical sense and balance of judgment to an extraordinarily fine degree. He was the perfect example of the ideal family doctor—a most trusted friend, unfailing in his help and understanding of personal problems. His stately bearing and kindly smile inspired thousands of his patients with supreme confidence and trust. He realized full well the inestimable value of a good family life and devoted all his energies to that end. He was, indeed, a revered husband, father, doctor, and friend. To his widow, two daughters, and three sons the deepest sympathy is extended by all who have been fortunate enough to have known this grand old man.

Dr. STEPHEN JOHN HENRY died suddenly at his home in Glasgow on July 20 at the age of 62. He studied medicine at Glasgow University and graduated M.B., Ch.B. in 1908. After holding resident hospital appointments he went into general practice in the Partick district of Glasgow in 1910. During the war of 1914–18 he served in France with the 70th Field Ambulance. He resumed general practice after the war and held several appointments, including membership of the Disabled Persons Advisory Panel in Glasgow. He was a former president of the Partick and District Medical Society. He leaves a widow, two sons, one of whom is a medical student, and two daughters.

Dr. HUGH COLLIN DAVIES died at his home in Colwyn Bay on July 29. He was born in Carmarthen in 1878 and was educated at University College, Cardiff, and Glasgow University. He graduated M.B., Ch.B. in 1903. He spent many years in practice in South and West Wales, and served in the R.A.M.C. in the first world war. He took his M.D.Glas. in 1925 and joined the staff of the Welsh Board of Health as Regional Medical Officer the same year. His long experience as a general practitioner enabled him to carry out his official duties in an outstanding way. Dr. Davies was known to medical practitioners throughout Wales as a kind friend, and patients referred to him were sure not only of sympathetic consideration but also of a first-class opinion. During the last war he was seconded by the Welsh Board of Health to the Emergency Hospital Service, and he became Hospital Officer for North Wales. As such he was responsible single-handed for organizing the emergency service throughout the six counties of North Wales. There is no doubt that the efforts he made during the war, which came at a time of his life when he was past his prime, must have contributed to the ill-health from which he suffered after his retirement in 1945. The sympathy of all who knew him will be extended to his widow.

Dr. GEORGE ANDREW, who was probably the oldest living Fellow of the Royal College of Surgeons, died at Brighton on July 29 at the age of 96. He was born in Devonshire and studied medicine at St. Bartholomew's Hospital. He qualified in 1874 and took his F.R.C.S. in 1879 and his M.D.Durh. in 1893. He practised in Egham, Gravesend, Torquay, and finally in Monte Carlo. To the end of his life he retained the full use of all his faculties, and he was renowned among his relations and friends for his amazing memory. He will be greatly missed by all who knew him.—L. G.

Dr. EDWARD LIONEL MACPHERSON RUSBY died suddenly at his home in London, S.W.2, on Aug. 1, at the age of 76. He was a student at King's College Hospital and qualified in 1896. He graduated M.B. two years later. After a short assistantship in general practice in Bromley he joined up with many other members of the Honourable Artillery Company as a private in the Civil Imperial Volunteers and served in the Boer War. He was later promoted Civil Surgeon in the South African Field Force. On his return to this country he settled in Brixton and built up a large practice as well as a quite considerable reputation as an obstetrician. He was honorary medical officer to the Water Lane Dispensary, district medical officer, and public vaccinator for Lambeth. This last appointment he retained until July 5 of this year. He was a member of the British Medical Association throughout the whole of his professional life, and for several years was honorary secretary of the Lambeth Division. He was a keen athlete in his younger days, and he led a full and active life until the morning of his death. His wife predeceased him by six months. He leaves a son and a daughter.

Dr. RICHARD HENRY WACE died in London on Aug. 5 at the age of 80. He studied medicine in Aberdeen and graduated M.B., C.M. in 1894. He had a varied medical career, having been coroner among the aborigines in the Northern Territory of Australia and medical officer in the Falkland Islands, where he did his round on horseback, once even covering a hundred miles in a single day. In spite of failing health he continued in active practice until he was 78. During the last war Dr. Wace turned earlier experience in a mental hospital to good effect by acting as locumtenent in mental hospitals. Dr. Wace was of distinguished appearance and his manner was charming and courtly. He dearly loved a classical allusion. He was proud of his 800-year-old descent from the Anglo-Norman poet Wace, or Eustace, of Jersey. He was a capable artist. He leaves a widow and a son.—E. S. S.

Medico-Legal

DAMAGES AGAINST A MEDICAL OFFICER OF HEALTH

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

A recent Irish case shows that when a medical officer of health, in attempting to protect the public against suspected carriers of typhoid, orders them to be discharged from employment he cannot be too careful to protect himself by observing all the regulations. Miss Annie O'Connor had been a cook on an Irish train. On June 29, 1944, a number of diners contracted typhoid fever, and Miss O'Connor herself caught the disease on July 21. She made a full recovery and was discharged cured. In March, 1945, she became assistant cook in the Grand Hotel, Greystones. In May a number of persons brought actions against the railway company but failed to establish negligence. On May 19, at the order of Dr. Gerald P. G. Beckett, medical officer of health of County Wicklow, she was discharged on suspicion of being a "precocious" typhoid carrier. (As was explained later, this word should have been "chronic" or "intermittent.") She sued him and the county council for negligence and breach of duty; an allegation of libel and slander against the doctor was

withdrawn.[1] The evidence showed that he made no tests when ordering Miss O'Connor to be discharged, and that afterwards three tests made at weekly intervals were negative. He did not carry out the regulation that in such a case a medical officer must give notice in writing to the employer that he considers it necessary that the suspect should submit to medical examination.

Miss O'Connor said in evidence that she was called to the private room of the manageress and asked by Dr. Beckett if she had had typhoid fever; she replied that she had, but had been discharged with a clean bill of health. Later the manageress sent for her and read out a letter from the doctor saying that she was possibly a precocious carrier of typhoid and must not on any account be employed in any position connected with the preparation or handling of food; she would therefore have to leave at once with a week's wages. The letter was a terrible shock to her. She took a post as a waiting-room attendant, and was offered one as a cook but was afraid to take it up. She was waiting for the case to clear her before she applied for another. No one associated with her or in contact with her had contracted typhoid. Miss Mary Loughnane, the manageress, gave evidence that Dr. Beckett, at his visit to her, did not seem to know what to do: he did not want to take the girl from employment, but at the same time wanted to do his duty. He rang up the hospital where the girl had been treated, and, judging by his answers, seemed to get no choice in the matter. He then said he was afraid she would have to let the girl go, and sent her the official letter.

Dr. C. J. McSweeney, chief medical officer of the fever hospital where Miss O'Connor had been a patient, said that before and after her discharge the usual tests were carried out and proved negative. That, however, did not necessarily mean that the outbreak on the train had not been due to a member of the staff. He had come to the tentative conclusion that the carrier had been Miss O'Connor. When Dr. Beckett asked him for information about Miss O'Connor, he said that it would be risky to keep the girl on without a check. Even a negative result would not enable anyone to certify her free from the danger of disseminating typhoid. She was an unsuitable person to employ as a cook. In the hospital she had had an unhealthy gall-bladder, and that predisposed to a carrier state. Women were five times as liable as men to become chronic carriers. It was difficult and tedious to clear a person conclusively of being a carrier; sixteen negative tests might be followed by a positive. Even now Miss O'Connor might be clear for three years and then give a positive result. The control of carriers was the hard core of the problem of prevention. A person undergoing test should not be employed in the preparation of food.

Professor J. W. Bigger, of Trinity College, Dublin, said that no examination at the hospital on the date of Miss O'Connor's discharge would have disclosed whether or not she was a chronic carrier, considering that it had taken three days with all the facilities of the laboratories to carry out the tests he had done in the case. The tests made at the hospital and after Miss O'Connor had left it were conventional but quite inadequate. In answer to the judge, he said that it would have been proper to give the girl warning when she left hospital, compensate her amply, and take precautions to see that she was never allowed to handle food until an investigation had been carried out. "In practice, it had better be a life sentence."

Dr. Beckett said in defence that when he received a letter from the medical officer of health for the city of Dublin about Miss O'Connor he considered that it required immediate action. Dr. McSweeney told him that an investigation had pointed to Miss O'Connor as the cause of the outbreak on the railway. He told Miss Loughnane that Miss O'Connor must not be employed in connexion with food, but he very definitely did not say that she must be sacked. He had no objection to her doing a job which did not involve the handling or preparation of food. There was no time for an examination; he felt it his primary duty to stop her from acting as a cook.

Dr. James Deeney, chief medical officer of the Department of Health, said it was a tribute to the public health officers that they had achieved a great reduction in the incidence of typhoid under practically unworkable and misleading regulations. The medical officer had practically no power. Dr. Beckett could not have prevented the girl from cooking without taking her to court.

Mr. Justice Maguire ruled that Dr. Beckett was for this purpose a statutory officer, responsible for the enforcement of the regulations to the Minister for Local Government and Public Health, and not the servant of the county council. Summing up for the jury, he said that no legal justification had been put forward for the dismissal of Miss O'Connor at the request of Dr. Beckett without warning. A strange feature of the case was that Miss O'Connor had been given no warning when she left hospital about the danger of her handling or preparing food. It also seemed a little strange that the public authorities responsible for the prevention of typhoid should not be a little more careful; possibly if they were they would have a little less trouble. Miss O'Connor had not been a party to the inquiry about the railway outbreak, and could not be bound by any decision taken at it. The jury found that Dr. Beckett had been negligent and awarded £750 damages.

[1] *Irish Press*, May 4–7.

Universities and Colleges

UNIVERSITY OF CAMBRIDGE

D. B. Cater, M.B., B.Chir., F.R.C.S., has been appointed to a University Demonstratorship in Pathology until Dec. 31, 1949; I. S. Longmuir, M.B., B.Chir., Assistant in Research in Colloid Science for two years from Oct. 1; and J. Marks, M.B., B.S., University Assistant Pathologist to Addenbrooke's Hospital for three years from Oct. 1.

The following medical degrees were conferred on Aug. 7:

M.D.—*D. Vérel, *F. A. Whitlock, R. R. Wilson, E. R. Hargreaves, R. B. Heisch, R. T. Hastings-James, H. I. Winner.

M.B., B.CHIR.—*L. Sefton, *D. I. Storey, *R. G. O. Tayler, *P. H. Abbott, *V. E. Amassian, *J. R. Bennett, *J. E. Forster, *W. M. Keynes, *W. F. W. Southwood, *J. D. Whitby, C. Q. Henriques, H. G. Ll. Lloyd-Thomas, *P. Allebone, *I. W. Broomhead, *E. J. Dowling, *C. P. E. Elliott-Binns, *D. G. Julian, *D. B. Sugden, *C. H. Talbot, *D. B. J. Wardle, D. S. Craig, W. J. D. Eberlie, A. A. McInnes, *R. A. Robinson, *J. G. H. Shaw, *K. R. Wallace, *R. D. Williams, *A. Ackroyd, *D. V. G. Feltham, *I. W. de G. Gregory, *N. Kennedy, *C. M. B. Pare, *H. Piggott, *W. R. Walsh, *A. R. H. Worssam, I. Henderson, P. F. D. Naylor, *H. I. Williams, *J. A. MacDougall, *G. E. W. Wolstenholme, *S. Powell, *J. E. S. Scott, *G. S. Smith, *E. A. Kauffmann, F. N. Hicks, P. J. Higgins, *J. L. Hine, *M. Honey, *E. F. Soothill, *J. Mander, *G. W. Page, *R. T. Sears, P. Fehrsen, C. C. K. Smith, *D. G. Bonham, R. A. Stanger, *P. G. Seed, *J. M. L. Shearer, J. C. Barker.

* By proxy.

UNIVERSITY OF GLASGOW

Thomas Ferguson Rodger, M.B., Ch.B.Glas., F.R.C.P.Ed., D.P.M., has been appointed to the recently founded Chair of Psychological Medicine in the University.

UNIVERSITY OF LONDON

Theodore Crawford, M.D., F.R.F.P.S., has been appointed to the University Chair of Pathology tenable at St. George's Hospital Medical School, from Oct. 1.

David Vaughan Davies, M.B., B.S., has been appointed to the University Chair of Anatomy tenable at St. Thomas's Hospital Medical School, from Oct. 1.

The following candidates have been approved at the examinations indicated:

M.D.—Branch I (*Medicine*): R. H. Andrews, A. Bogdanovitch, H. J. Boutourline-Young, R. E. Bowers, E. J. S. N. Briggs, R. N. Cates, W. E. Clarke, C. F. Cooper, P. G. Dalgleish (*University Medal*), A. St. J. Dixon, M. Elias, R. H. J. Fanthorpe, D. A. Ferguson, A. Freedman, T. J. B. Geffen, J. H. Goonewardene, J. L. Greaves, D. B. Irwin, H. B. Kelly, R. E. Kelly, R. King-Brown, V. M. Leveaux, I. P. McL. MacDougall, G. MacGregor, R. I. Meanock, J. B. Mehta, D. E. Meredith, J. B. Mitchell, G. Monckton, P. B. Newcomb, T. M. Ll. Price, K. O. Rawlings, T. L. Reeves, G. K. C. Rettie, Elizabeth V. Rohr, J. Rubie, J. P. Shillingford, Honour M. V. Smith, J. S. Staffurth (*Mark of Distinction*), C. Symons, R. B. Terry, F. J. H. Walters, H. F. West, A. A. Williams, G. F. Willson. Branch II (*Pathology*): R. D. Clay, E. A. Fairburn, R. C. B. Pugh, R. W. Riddell, K. A. D. Turk, P. T. J. C. P. Warner (*University Medal*). Branch IV (*Midwifery and Diseases of Women*): Lois E. Hurter, F. L. E. Musgrove. Branch V (*Hygiene*): Patricia M. Elliott, J. Knowelden, P. A. B. Raffle, A. T. Roden, I. Taylor, P. Tomlinson, P. A. Tyser, W. A. Wilson. Branch VI (*Tropical Medicine*): A. J. Duggan, J. T. Harold, J. M. Wilson.

ROYAL COLLEGE OF SURGEONS OF ENGLAND

We are asked to state that the Members of the Board of the Faculty of Anaesthetists, whose names were announced in this column last week (p. 359), have also received the Fellowship of the Faculty (F.F.A., R.C.S.).

No. 31

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended July 31.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1948					1947 (Corresponding Week)				
	(a)	(b)	(c)	(d)	(e)	(a)	(b)	(c)	(d)	(e)
Cerebrospinal fever	40	2	17	4	—	48	11	17	2	1
Deaths		—	—				—	2		
Diphtheria	125	15	28	18	5	200	26	29	16	6
Deaths	2	—	—	—	—	2	—	—	1	1
Dysentery	77	24	35	—	—	53	9	10	—	—
Deaths				—	—				—	—
Encephalitis lethargica, acute	2	—	2	—	—	2	—	—	—	—
Deaths							—			
Erysipelas			33	6	—			22	8	2
Deaths		—					—			
Infective enteritis or diarrhoea under 2 years				18					52	
Deaths	40	2	2	3	1	52	4	17	1	1
Measles*	8,211	457	59	87	59	6,954	366	61	336	8
Deaths†			—	—	1	1	—	—	—	—
Ophthalmia neonatorum	54	4	10	1	—	80	7	19	3	1
Deaths										
Paratyphoid fever	35	1	2(B)	—	—	19	—	1(A)	1(B)	—
Deaths	—	—	—	—	—	—	—	—	—	—
Pneumonia, influenzal	355	23	2	3	1	271	17	1	—	3
Deaths (from influenza)‡	4	1	1	—	—	3	—	—	—	—
Pneumonia, primary			93	29				79	17	
Deaths	195	31		10	6		20			6
Polio-encephalitis, acute	1	—				39	7			
Deaths							—			
Poliomyelitis, acute	38	6	4	—	1	448	53	37	5	11
Deaths§	2	—					3			
Puerperal fever		1	5		2		—	9		—
Deaths										
Puerperal pyrexia‖	122	9	8	1	1	145	14	6	—	3
Deaths		—					—			
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,329	83	208	64	18	803	80	98	32	20
Deaths†			—	—	—	—	—	—	—	—
Smallpox	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Typhoid fever	9	—	1	1	—	6	—	4	3	2
Deaths	—	—	—	—	—	—	—	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Whooping-cough*	3,309	275	8	78	10	1,737	195	29	71	5
Deaths	8	—	—	1	1	8	2	—	3	3
Deaths (0–1 year)	297	23	29	13	11	320	45	67	31	7
Infant mortality rate (per 1,000 live births)										
Deaths (excluding still-births)	4,830	781	587	175	107	3,900	609	530	184	101
Annual death rate (per 1,000 persons living)			11·8	10·9				11·0		
Live births	8,011	1230	913	409	228	8,972	1404	1073	365	251
Annual rate per 1,000 persons living			18·4	25·6				21·6		
Stillbirths	211	22	39			252	32	40		
Rate per 1,000 total births (including stillborn)			41					36		

* Measles and whooping-cough are not notifiable in Scotland, and the returns are therefore an approximation only.

† Deaths from measles and scarlet fever for England and Wales, London (administrative county), will no longer be published.

‡ Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

§ The number of deaths from poliomyelitis and polio-encephalitis for England and Wales, London (administrative county), are combined.

‖ Includes puerperal fever for England and Wales and Eire.

EPIDEMIOLOGICAL NOTES

Discussion of Table

In *England and Wales* infectious diseases were less prevalent during the week. There was a decrease in the incidence of scarlet fever 418, measles 290, whooping-cough 50, acute pneumonia 30, dysentery 31, and diphtheria 14, while an increase was recorded in the notifications of paratyphoid fever 26 and cerebrospinal fever 15.

A fall in the notifications of scarlet fever occurred throughout the country; the largest fall was 75 in Lancashire. The largest decreases in the incidence of measles were London 95, Durham 81, and Essex 73; the largest rises were Yorkshire West Riding 192 and Gloucestershire 67. The chief variations in the returns for whooping-cough were falls in Middlesex 83, Yorkshire West Riding 55, and Durham 53, with rises in Cheshire 56 and Sussex 42.

The notifications of diphtheria (125) reached the lowest level yet recorded and were 9 below the preceding lowest level. The largest variations in the returns for diphtheria during the week were a decrease of 10 in Yorkshire West Riding and an increase of 9 in Cheshire.

The largest returns for dysentery were London 24, Lancashire 11, and Yorkshire West Riding 10. Paratyphoid fever reached the highest level for almost a year; the two principal centres of infection were Northamptonshire 14 (Corby U.D. 10) and Sussex, Eastbourne C.B. 10.

Notifications of acute poliomyelitis have been practically constant during the past three weeks. Multiple cases were notified during the week from London 6 (Westminster 4); Middlesex 5 (Heston and Isleworth U.D. 2, Wood Green M.B. 2); Yorkshire West Riding 4; Yorkshire East Riding 3 (Kingston-upon-Hull C.B. 2); Southampton 3 (Southampton C.B. 2); Lancashire 3; Warwickshire 2; and Yorkshire North Riding 2.

In *Scotland* a decreased incidence was recorded for scarlet fever 37, acute primary pneumonia 29, dysentery 17, and measles 11. Of the 35 cases of dysentery 25 were notified in Glasgow. Notifications of cerebrospinal fever increased from 10 to 17, and of these cases 14 were notified in Glasgow.

In *Eire* a fall occurred in the notifications of whooping-cough 21, measles 16, and diarrhoea and enteritis 12. The largest outbreak of measles during the week was at Monaghan, Castleblayney R.D. 30.

In *Northern Ireland* the only fluctuation in the trends of infectious diseases was a decrease of 23 in the notifications of scarlet fever.

Quarterly Returns for England and Wales

During the first quarter of the year the births were equivalent to a rate of 18.9 per 1,000, which was 3.9 below the rate for the March quarter of 1947. Infant mortality was 41 per 1,000 live births and was the lowest rate ever recorded in a first quarter, being 11 below the rate for the first quarter of 1947 and 25 below the average rate for the March quarters of the 10 preceding years, 1937–46. The general death rate was 12.4 per 1,000, the lowest rate ever recorded for a March quarter. It was 4.7 below the rate for the first quarter of 1947 and 1.9 below the average of the first quarters of the five years 1942–6. Only 644 deaths were attributed to influenza; the lowest number previously recorded in a first quarter was 1,470 in 1945. The ten-yearly average of influenza deaths for the first quarters of 1938–47 was 3,800.

Quarterly Returns for Northern Ireland

During the first quarter the birth rate was 22.1 per 1,000, which was 1.2 below the average rate for the five preceding first quarters. Infant mortality was 53 per 1,000 registered births compared with an average rate of 78 for the corresponding quarters of the five preceding years. Maternal mortality was 2.4 per 1,000 births and was 0.5 below the five-year average. The general death rate was 12.8 per 1,000 and was 3.3 below the average rate for the first quarters of the five preceding years. Deaths attributed to the principal epidemic diseases numbered 92, and included 43 deaths from influenza, 26 from diarrhoea and enteritis in children under 2 years, and 16 from whooping-cough. Deaths from pulmonary tuberculosis numbered 207, and there were 44 deaths from other forms of tuberculosis. These totals were 39 and 30, respectively, below the average of the first quarters of 1943–7.

Week Ending August 7

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 848, whooping-cough 3,185, diphtheria 107, measles 6,879, acute pneumonia 363, cerebrospinal fever 40, acute poliomyelitis 38, dysentery 111, paratyphoid 47, and typhoid 10.

Medical News

Tuberculosis in Scotland

The Secretary of State for Scotland has asked the Scottish Health Services Council to assist him in making a special examination of the tuberculosis position in Scotland, and the Council has set up a special committee for this purpose. The committee includes three members of the Council—Captain J. P. Younger, of Alloa, who is chairman of the committee; Dr. Matthew Fyfe, Medical Officer of Health of Fife County; and Dr. J. R. Langmuir, a general practitioner in Glasgow. The other five members of the committee are Professor Cameron, Professor of Tuberculosis in Edinburgh University; Professor Crew, Professor of Public Health in Edinburgh University; Dr. H. C. Elder, who is in charge of mass radiography in Edinburgh; Dr. S. I. A. Laidlaw, medical officer of health of Glasgow; and Dr. Maclean, who is in charge of mass radiography arrangements in Glasgow.

British Tuberculosis Association

At its recent annual general meeting the Tuberculosis Association decided to change its name to the British Tuberculosis Association.

Waste Straw

Professor S. Zuckerman, of Birmingham University, has been appointed head of a Government committee to find a use for waste straw.

Sir Harold Gillies

The King of Norway has conferred the decoration of Commander of the Order of St. Olav upon Sir Harold Gillies in recognition of services rendered during the war.

Psychotherapeutic Conference

From Aug. 3 to 9 psychiatrists and psychologists from Australia, Chile, China, Denmark, Finland, France, Holland, Norway, Sweden, Switzerland, South Africa, the U.S.A., and Great Britain took part in an international technical psychotherapeutic conference at the Institute of Child Psychology; this was complementary to the International Congress on Mental Health. At the close of the conference the possibilities were discussed of retaining the links formed at the conference between child psychiatrists and psychologists in an informal international association of national groups.

Tribute to Pharmaceutical Society

The Minister of Health paid a tribute to the Pharmaceutical Society when he was the Council's guest at their monthly dinner on July 27. They had always got on well together, he said, and it was a fact that the discussions with the pharmaceutical profession had been more fruitful than with any other branch of the medical world. This great scheme had many tributaries, and the pharmaceutical was not the least of them. It would be almost impossible to launch a ship of the size of the National Health Service without considerable water displacement. Other countries were watching us. Britain had not been exhausted by 6¼ years of war; we had been inspired by the war and our energies had hardly been tapped.

Wills

Sir George Newman, of Grims Wood, Harrow Weald, late Chief Medical Officer of the Ministry of Health, left £58,618. Professor Henry Arthur Burgess, of Cheadle, Cheshire, President of the B.M.A. in 1929 and for many years Professor of Clinical Surgery in Manchester University, left £122,276 3s. 9d. Dr. Henry Willoughby Gardner, of Church Stretton, Salop, left £68,120 16s. 6d.

COMING EVENTS

Physical Medicine

A short course of lectures on the various aspects of physical medicine has been arranged on Tuesdays and Thursdays at 5 p.m. from Sept. 2 to Oct. 7 inclusive, and will be suitable for candidates preparing for Part II of the Diploma in Physical Medicine. Further details can be obtained from the honorary secretary, British Association of Physical Medicine, 45, Lincoln's Inn Fields, London, W.C.2.

Conference on Infertility

The annual conference on infertility, organized by the Family Planning Association (69, Eccleston Square, London, S.W.1), will be held at Exeter on Saturday and Sunday, Sept. 25 and 26. A draft programme has been arranged as follows: Sept. 25, 10 a.m., "Congenital Absence of the Vas," by Mr. D. Young, and "Application of the Supravital Stain to Human Spermatozoa," by Dr. A. C. Crooke and Miss Anita Mandl; 2.15 p.m., "Some Lessons from 3,000 Utero-tubal Insufflations," by Dr. Albert Sharman; "Hysterosalpingography in Water Soluble Radio-opaque Media," by Miss Josephine Barnes; and "Vitamin E Therapy for Sub-fertility in the Male," by Dr. M. Hadley Jackson and Dr. H. A. Davidson. Sept. 26, 10 a.m., contributions by members of the Royal Free Hospital Sub-fertility Unit. Introduction by Dr. Gertrude Dearnley; "The Value of the Sim's Test in the Diagnosis of Male Infertility," by Dr. Mary Barton; "Report on a Series of Testicular Biopsies," by Dr. E. Friedman; "Results of a Series of Testosterone Implants," by Miss M. Moore White; "Preliminary Investigation in the Treatment of Oligozoospermia by Vitamin E," by Mr. Norman Warren; "The Blood Supply of the Testis and its Relation to Sterility," by Dr. R. G. Harrison; and "The Effect of Dilution on Sea Urchin Spermatozoa," by Lord Rothschild; 2.15 p.m., "The Critical Assessment of Infertility Data," by Dr. G. I. M. Swyer; "Coitus and Infertility in the Rabbit," by Dr. Harris; "Relationship between Miscarriage and Seminal Characteristics as demonstrated by Seminal Analysis," by Mrs. Harvey.

SOCIETIES AND LECTURES

Friday

EDINBURGH POSTGRADUATE BOARD FOR MEDICINE.—At Anatomy Lecture Theatre, Edinburgh University, Aug. 27, 3.30 p.m. "*Present State of Chemotherapy in Cancer*," by Professor A. Haddow.

APPOINTMENTS

BECK, DIANA J. KINLOCH, M.B., B.S., F.R.C.S., Neuro-surgeon, Middlesex Hospital, London, W.

JONES, D. J., M.B., B.Ch., D.P.H., Medical Officer of Health for Rugby Borough and Rural District and Area Medical Officer.

MILLAR, I. B., M.D., D.P.H., Assistant Medical Officer, Newry Division, County Down, Northern Ireland.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Cleland.—On Aug. 3, 1948, at the Ayrshire Central Hospital, Irvine, to Joan, wife of Mr. Gavin Cleland, F.R.C.S.Ed., a daughter.

Cowper.—On Aug. 4, 1948, at the Anglo-American Hospital, Cairo, to Grace (née Boxley), wife of Dr. S. G. Cowper, a daughter—Ann Christine.

Kennedy.—On Aug. 8, 1948, at Queen Charlotte's Hospital, to Minnie, wife of Dr. Gordon Kennedy, of Twickenham, a daughter—Ester Margaret.

Paget Davis.—On Aug. 12, 1948, at St. John's Hospital, Chelmsford, to Lilian (née Vaughan), wife of Dr. Donald Paget Davis, of Braintree, Essex, a second son—Mark Scott.

Pearsall.—On Aug. 7, 1948, to Mary, wife of Dr. P. R. Pearsall, Mill Hill, London, N.W. a daughter.

Pearson.—On Aug. 13, 1948, at St. Brenda's Nursing Home, Bristol, to Elise (née Powell), late Q.A.R.N.N.S.(R.), wife of Dr. G. M. Pearson, a daughter, prematurely—Fleur.

White.—On Aug. 8, 1948, at Selly Oak Hospital, Birmingham, to Edna, wife of Mr. J. R. A. White, F.R.C.S., a daughter.

MARRIAGES

Gillis—Greaves.—On Aug. 12, 1948, at St. John's Wood Synagogue, London, Leon Gillis, M.B.E., M.B., B.Ch.Orth., F.R.C.S., to Rachel Greaves, of Sheffield.

Kendall—Morgan.—On June 26, 1948, at Huntington, Kington, Herefordshire, Arthur C. Kendall, M.R.C.P., to Hilda M. Morgan, M.B., Ch.B.

DEATHS

Arnold.—On Aug. 6, 1948, at Worthing, Miles Bracewell Arnold, M.D., D.P.H., late of the Ministry of Health.

Borrowman.—Recently, Alexander Paterson Robertson Borrowman, M.B., Ch.B., of Bramptoc, Cumberland, aged 45.

Brice.—On Aug. 7, 1948, at Thornhill Edge, Dewsbury, Yorks, Henry Doyle Brice, M.R.C.S., L.R.C.P., aged 74.

Hounsfield.—Recently, Maurice Coupland Hounsfield, M.B., B.Chir., of Stoke Bishop, Bristol, aged 43.

Mackie.—On Aug. 8, 1948, while bathing at Sandown, Kenneth William Mackie, M.R.C.S., L.R.C.P., of Salisbury Green, Southampton, aged 44.

Maitland.—On Aug. 10, 1948, Thomas Gwynne Maitland, M.D., of Hatchmere Wood, Norley, Cheshire, and late of the Cunard White Star Line.

Murray.—Recently, while on holiday at Dornoch, David Alexander Murray, M.B., Ch.B.Ed., of Helmsley, Yorks.

Nimmons.—On Aug. 6, 1948, Robert Nimmons, M.B., B.Ch., of Wheldon House, Pelton, Co. Durham.

Reckless.—On Aug. 15, 1948, at High Lane, near Stockport, Cheshire, Philip Alfred Reckless, F.R.C.S., aged 65.

Sutherland.—On Aug. 14, 1948, at 3, Darnaway Street, Edinburgh, Francis Benjamin Sutherland, M.B., Ch.B., D.P.H.Ed.

Walker.—Recently, Henry Francis Bell Walker, M.D., of Cape Colony, South Africa, aged 72.

Williams.—On Aug. 9, 1948, at Oakmead, Exmouth, S. Devon, Percy Glyn Savours Williams, M.D., late of Cairo.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Suppression and Treatment of Malaria

Q.—*What is the value of "paludrine" therapeutically and as a suppressive? Can any order of preference be given as between paludrine, mepacrine, and quinine for both purposes?*

A.—Paludrine is a valuable drug in the treatment of malarial infections due both to *Plasmodium vivax* and to *Plasmodium falciparum* owing to its lack of toxicity. In acute attacks due to *P. vivax* it will control fever and parasitaemia but will not prevent a subsequent relapse. In acute attacks due to African strains of *P. falciparum* it is slightly slower in its action than either mepacrine or quinine. It will not eradicate *P. falciparum* infections as will mepacrine, and it is not a true causal prophylactic either for *P. vivax* or for all strains of *P. falciparum*. Paludrine is possibly less active than mepacrine or quinine against *P. malariae*.

It is now recognized that paludrine, mepacrine, and quinine have essentially the same action on malaria parasites. In the control of an acute attack paludrine is to be preferred owing to its lack of toxicity. Mepacrine stains the skin and may cause gastro-intestinal discomfort and, in very rare instances, skin lesions and psychotic symptoms. Quinine may cause cinchonism, buzzing in the ears, or deafness. If the infection is due to *P. falciparum* or if *P. falciparum* is the prevailing parasite in the area quinine must be avoided both in treatment and in suppression because of the danger of inducing blackwater fever. If a vivax infection is to be eradicated recourse must be had to quinine and pamaquin or to quinine and pentaquine; if a falciparum infection is to be eradicated mepacrine will be effective; neither quinine nor paludrine alone will eradicate either *P. falciparum* or *P. vivax* infections. For suppression, the drug chosen must be taken daily. Mepacrine is a better suppressive than quinine for all forms of malaria. Results of controlled investigations into the comparative efficiency of mepacrine and paludrine as suppressives are not at present available.

Agenized Flour

Q.—*What is "agenized" flour and why has it been adopted for general use both by ourselves and by the Americans?*

A.—"Agene" is the trade name for nitrogen trichloride, which in the pure state is a very unstable compound. It is prepared commercially in impure form by passing chlorine into an aqueous solution of an ammonium salt. A current of air containing about 1% of agene is then passed into the flour, with adequate agitation, until a sufficient quantity has been absorbed. Agene has a strong bleaching action, but it also "improves" the flour so as to give a tough tenacious dough which bakes into a large porous loaf. Its trade name suggests its function in artificially ageing and maturing the flour. Reasons for the use of agene in preference to other bleaching or improving agents presumably lie in the ease and cheapness of its industrial application and in its efficiency in producing the desired properties in the flour.

Tuberculous Rheumatism

Q.—*Is the condition of tuberculous rheumatism a definite entity and, if so, how is the diagnosis established? A boy of 15 had a febrile attack with pain in one knee. Overnight he developed a bilateral effusion in both knees which was painless. Simultaneously he developed erythema nodosum. His temperature fell after three days (he was on salicylates), and the Mantoux reaction was positive in a dilution of 1 in 10,000. No other joints were affected. There is no contact history of tuberculosis. Could this be a case of tuberculous rheumatism, and, if so, is treatment on sanatorium lines indicated?*

A.—In 1902 Poncet described a form of polyarthritis which he believed to be tuberculous, and the "Poncet" type of rheumatoid arthritis is definitely recognized in France. Reitter and Löwenstein claimed to have isolated the tubercle bacillus in the blood stream in these cases by a special technique, but others have failed to obtain the same results. Brav and Hench (*J. Bone Jt Surg.*, 1934, **16**, 839), after careful study, doubted the existence of a tuberculous form. Copeman (*Reports on Chronic Rheumatic Diseases*, 1936, **2**, 24) discussed the subject very fully and described cases which appeared to be tuberculous. On the whole it seems probable that the tubercle bacillus may be responsible for some cases of polyarthritis of the rheumatoid type.

The case described in the question does not, however, appear to be of this character, and such a diagnosis could not safely be reached at this stage. Careful observation over a long period would be required, and the first step should be an exhaustive search for any possible tuberculous focus in the body, possibly glandular. At a later stage, if the signs persisted, joint biopsy might be justified. The positive Mantoux reaction might be due to a healed focus, possibly calcified glands, without necessarily being associated with the joint attack. Erythema nodosum may be tuberculous or streptococcal in origin, or may occur without evidence of either, though a tuberculous infection is believed to be a predisposing factor. Subacute rheumatism may be a possible diagnosis, though the painlessness of the effusion is against this; some of the cases recorded as tuberculous rheumatism began with symptoms suggestive of subacute rheumatism. The general character of the attack is more like an intermittent hydrarthrosis, perhaps caused by some allergic process. The age of onset and the character of the joint symptoms are against rheumatoid arthritis, though such a diagnosis could not be entirely excluded. Treatment might reasonably be carried out on sanatorium lines, that is, by rest, fresh air, cautiously graduated heliotherapy, and tonic measures, but not in a sanatorium—a sea-bathing hospital would be more appropriate.

Treatment with Vitamin B_2

Q.—*I have had a case of scrotal eczema with pruritus ani. The eczema was cured and the pruritus controlled by vitamin B_2 (riboflavin, 3 mg. morning and night), but treatment causes nasal congestion. Can you explain this, suggest a remedy, and say if it is dangerous to increase the dose?*

A.—It is very unlikely that scrotal eczema and pruritus ani would respond to treatment with riboflavin unless the condition is due to a gross vitamin-B-complex deficiency. In this case treatment with a vitamin-B-complex preparation such as yeast, liver, or "marmite" is likely to be more effective than with any single vitamin. It is also very unlikely that the riboflavin causes nasal congestion. It is an extremely harmless substance, no side-effects from its use have been reported, and no harm can come by increasing the dose—although this would appear to be unnecessary, since 6 mg. a day, the dose given by the inquirer, is quite enough for the treatment of mild deficiency.

Reversion of Changes caused by Androgens

Q.—*Are the changes brought about by the administration of androgens reversible? The case I have in mind is that of a young woman who was treated abroad with large doses of male hormone. She now has a pronounced enlargement of the clitoris and other signs of virilism. Can any treatment be suggested?*

A.—The changes are largely reversible, but the reversion may take several months. In the case of the clitoris and the larynx complete reversion to the condition existing before androgen therapy was begun does not necessarily occur. There is no hormone treatment likely to counteract the residual changes.

Treatment of Varicose Veins after Sympathectomy

Q.—*A patient has developed a small varicose vein in the leg, which should be injected with some sclerosing agent. Is the fact that a sympathectomy was successfully performed five years ago for erythrocyanosis cruris puellarum a contraindication to injection treatment? The circulation in the legs has been quite good since the operation.*

A.—There is no objection at all to the injection of the veins in erythrocyanosis cruris puellarum frigida whether sympathectomy has been done or not. Varicose veins are often associated with this condition, and their treatment helps it slightly.

Ulceration of Mucous Membranes

Q.—*A mother of three children, aged 42, has suffered continuously for 18 years from multiple small ulcers on the mucous membrane of the mouth, tongue, and vagina. They started after her first confinement and became chronic after her second. Sometimes she has only two or three, on one occasion she had 19. She once had haemorrhoids, and the ulcers appeared on the extruded mucous membrane. The Wassermann reaction is negative, test-meal examination revealed nothing abnormal, and the blood and urine are normal. Ultra-violet radiation improved the vaginal ulcers, but mouth-washes aggravated the mouth condition. Culture from the ulcers gave growth of staphylococci and streptococci, which were only mildly sensitive to penicillin. What would you suggest for a cure?*

A.—Coincident ulceration of the mouth, vulva, vagina, and sometimes anus, though not very common, is a syndrome well known if only because its treatment is so unsatisfactory. The relation of ulceration to menstruation is not mentioned in the question, but very often it is striking; nor is it stated whether the ulceration cleared up during pregnancy, which it usually does. Many questions have been received on similar cases, and replies published in the *Journal* (March 15, 1947, p. 365; June 15, 1946, p. 940; Dec. 25, 1943, p. 839) should be consulted. The cause of the condition is unknown, although there are many theories to account for it. Simultaneous affection of the vulva and mouth is a feature of several diseases—e.g., glossitis and vulvitis in diabetes and in various anaemias; angular stomatitis and pruritus vulvae in anaemias, ariboflavinosis, and other deficiency states. The typical cyclical ulcerations may have an endocrine cause, and a recent suggestion put forward by B. Zondek and Y. M. Bromberg (*J. Obstet. Gynec. Brit. Emp.*, 1947, **54**, 1) is that the condition is sometimes an allergic manifestation to one or other hormone. They give details of tests which can be applied. Previous writers have pointed out that there is sometimes a strong family history of various allergic states in these cases. The patient should be carefully investigated for evidence of dietetic deficiency, anaemia, and diabetes, and skin-sensitivity tests should be carried out. Any focus of infection in the mouth should be dealt with, as should any form of dyspepsia. If no obvious cause is found, empirical treatment with large doses of vitamins A, B, and C should be tried in turn. Chorionic gonadotrophin, 500 i.u. intramuscularly twice weekly, appears to help in some cases.

Low-calorie Diet for Obesity

Q.—*Can you give details of a diet for obesity, supplying 1,000 calories daily, having regard to present-day difficulties of supply and to the patient's dislike of cooked green vegetables—e.g., cabbage, cauliflower, sprouts? The patient eats lettuce and other salad vegetables, also peas, French beans, carrots, and beetroot.*

A.—The following diet will meet most of the requirements of the questioner. Difficulties arise when attempts are made to give the patient a sensation of satiety. This has usually been done by distending the stomach with green vegetables and fruit, which have a negligible calorie value. It is questionable whether this is the correct course; the fundamental aim in treating obesity is to educate the patient to control his appetite, and it is better that he should suffer for a few days the pangs of unassuaged hunger than to pander to his lust for repletion. The intense hunger disappears after a week, and during this time it is perhaps justifiable to keep it in check with "benzedrine"; thereafter a low-bulk diet can be taken without distress.

Breakfast.—Raw fruit, no sugar; one boiled or poached egg, or grilled kidney, or smoked haddock, or steamed white fish; two thin slices of bread, toasted if desired, butter, jam, or marmalade very thinly spread; tea or coffee with milk to taste.

Lunch.—Clear soup; lean meat, chicken, rabbit, boiled white fish, lobster, crab, as much as desired; salad as much as desired; fruit, raw or cooked without sugar, as much as desired.

Tea.—Two thin slices of bread very thinly buttered, with tomato, cucumber, cress, or "marmite" as a sandwich filling; tea with milk to taste, no sugar.

Dinner.—As for lunch.

A concentrate of vitamins A and D should be prescribed.

Yellow-fever Immunization

Q.—*What is the most satisfactory method of yellow-fever immunization? Is it now required that a second inoculation be given six months after the first, with annual boosters thereafter?*

A.—This is not the usual ruling. Certificates of yellow-fever vaccination as were recognized by Unrra and generally accepted the world over are valid for four years. Individuals in the midst of an epidemic would probably be wise to be reinoculated after a shorter interval. The accepted vaccines are those made from chick embryos infected with the attenuated 17D strain, which is made in North and South America, England, and South Africa, and the mouse-brain vaccine made in Dakar from mice infected with the French neurotropic strain. A single injection of vaccine subcutaneously is given in either instance.

Death Rate from Cancer

Q.—*As medical officer of health to a population of 8,900 in Scotland I should like to know if our 16 deaths from cancer is higher or lower than the rate for England and Wales?*

A.—The age constitution of the population at risk is an important factor in comparing death rates from cancer, since this disease is one of later life. If the age structure of the population was proportionately the same as in England and Wales then, on the basis of the mortality experience of England and Wales, the number of expected deaths would be 17.

Use of Tap-water for Injections

Q.—*Are there any objections to using London tap-water in place of distilled water for the usual subcutaneous and intramuscular injections?*

A.—In an emergency, and when distilled water is not available, tap-water may be used for dissolving a tablet of, say, an alkaloid to be given by hypodermic injection. How far this practice should be extended depends on the nature of the medicament and the volume to be injected. London water is chlorinated and also contains various mineral constituents, mainly salts of calcium and magnesium; anything reacting with these substances should naturally not be made up in tap-water. The injection of a considerable volume would be inadvisable, owing to the possible presence of pyrogens; these are more likely to be present if the water comes through a cistern which needs cleaning. The injection of any solution made from water which has not been autoclaved involves a very small risk of producing gas-gangrene owing to the chance presence of spores of *Clostridium welchii* or some related organism. For this reason alone the practice should be discountenanced.

NOTES AND COMMENTS

British Rheumatic Association.—Dr. F. HERNAMAN-JOHNSON, Chairman of the Council of the British Rheumatic Association, writes: As you rightly say in your report (June 26, p. 1249), the British Rheumatic Association is chiefly a lay body. It leaves matters of research strictly to the Empire Rheumatism Council, but it is directly interested in securing that treatment based on existing knowledge shall be readily available to all rheumatic sufferers. We would welcome a large number of general practitioner members. They could tell us the needs of their particular district and help to increase the membership of the association among their patients. A very large total membership is aimed at, perhaps 100,000, and this can be reached only by the co-operation of that part of the medical profession which comes into intimate contact with the people. The literature of the British Rheumatic Association will be sent to anyone who cares to write to 5, Tite Street, Chelsea, London, S.W.1.

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